In the United States Court of Federal Claims

OFFICE OF SPECIAL MASTERS

Filed: April 7, 2016

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ERICA FESTER, parent of B.A.B., a minor,	
Petitioner,	No. 10-243V
v.	Chief Special Master Dorsey
SECRETARY OF HEALTH AND HUMAN SERVICES,	Proquad ("MMRV") Vaccine; Autism ("ASD"); Encephalopathy; Subacute Encephalopathy; Subclinical Encephalopathy;
Respondent.	Insufficient Proof.
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Peter Joseph Sarda, Creech Law Firm, Raleigh, NC, for petitioner.
Voris Edward Johnson, U.S. Department of Justice, Washington, D.C., for respondent.

DECISION ON PETITIONER'S MOTION FOR RULING ON THE RECORD[1]

I. Introduction

On April 15, 2010, Erica Fester ("petitioner"), parent of B.A.B., a minor, filed a petition for compensation under the National Vaccine Injury Compensation Program ("the Program"),[2] alleging that the combined measles, mumps, rubella, and varicella ("MMRV" or "Proquad") vaccine[3] that B.A.B. received on April 18, 2007, caused him to suffer encephalopathy. Petition

[1] Because this decision contains a reasoned explanation for the undersigned's action in this case, the undersigned intends to post this ruling on the website of the United States Court of Federal Claims, in accordance with the E-Government Act of 2002, 44 U.S.C. § 3501 note (2012)(Federal Management and Promotion of Electronic Government Services). As provided by Vaccine Rule 18(b), each party has 14 days within which to request redaction "of any information furnished by that party: (1) that is a trade secret or commercial or financial in substance and is privileged or confidential; or (2) that includes medical files or similar files, the disclosure of which would constitute a clearly unwarranted invasion of privacy." Vaccine Rule 18(b).

[2] The Program comprises Part 2 of the National Childhood Vaccine Injury Act of 1986, 42 U.S.C. §§ 300aa-10 et seq. (hereinafter "Vaccine Act" or "the Act"). Hereafter, individual section references will be to 42 U.S.C. § 300aa of the Act.

[3] B.A.B. received one dose of the vaccine Proquad, which contains the measles, mumps, rubella,

at ¶ 5. Petitioner contends that B.A.B.'s "loss of prior skills and his current developmental delay is [sic] the sequela of that brain injury." Id. The medical records and other information in the record, however, do not support a finding that petitioner is entitled to compensation.

Under the Program, petitioner may not receive compensation based solely upon her claims, as the petition must be supported by either medical records or by the opinion of a qualified physician proving a causal relationship. See § 13(a)(1). Here, the medical records do not support petitioner's claims, so a medical opinion is required. Petitioner has offered the opinion of Dr. Karen Harum.[4] However, Dr. Harum's opinion fails to provide support for the elements necessary to prove causation.

For these reasons, and the reasons discussed below, petitioner has failed to demonstrate that she is entitled to compensation.

I. Procedural History

Petitioner filed her case *pro se* on April 15, 2010. In support of her claim, petitioner filed medical records labeled as exhibits 1-12. Approximately three months later, she filed additional medical records and videotapes. See Pet'r's Exs. 16-17 filed Aug. 19, 2010 (ECF Nos. 10).[5]

On September 10, 2010, respondent filed her Rule 4(c) Report, stating that the case was not appropriate for compensation. Petitioner filed a response to the Rule 4(c) Report on February 8, 2011. Although petitioner argued that three of B.A.B.'s treating doctors believed B.A.B. suffered an encephalopathy and two believed that B.A.B. "had received a vaccine injury," she agreed that she needed the report of a medical expert to support her claim. Pet'r's Response to Respondent's Rule 4(c) Report at 3-4 (ECF No. 20).[6]

Because B.A.B. was diagnosed with autism by at least one treating doctor, the case was reassigned, over petitioner's objection,[7] to former Chief Special Master Vowell, one of the

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and varicella vaccines. Petitioner's Exhibits ("Pet'r's Exs.") 3 at 2-3; 6 at 15.

[4] See Pet'r's Exs. 19, 26, 29 (initial and two supplemental reports). Dr. Harum is a neurodevelopmental pediatrician who began treating B.A.B. in September 2008. See Respondent's Exhibit ("Resp's Ex.") A at 1 (ECF No. 27) (medical records from Dr. Harum). Because the documents submitted as Ex. A were not electronically filed and do not have ECF numbers, the undersigned refers to the handwritten page numbers on the bottom of the document.

[5] Exhibit 16 is comprised of several photographs and movies of B.A.B as a young child. Exhibit 17 is a video clip of B.A.B. saying, "I love you."

[6] The pages of Petitioner's Response to Respondent's Rule 4(c) Report are unnumbered, but this quotation appears on the bottom of the third and the top of the fourth pages of the document.

[7] See Motion dated Feb. 23, 2011 (ECF No. 18). See also Order dated Feb. 28, 2011 (ECF No. 19).

special masters involved in the Omnibus Autism Proceeding ("OAP").[8] During this time, petitioner continued to file medical records. See Pet'r's Ex. 18 filed Apr. 14, 2011. Because petitioner experienced difficulty obtaining the medical records from Dr. Harum, one of B.A.B.'s treating physicians, respondent was authorized to issue a subpoena and then obtained and filed Dr. Harum's records on June 17, 2011. See Resp's Ex. A; Order dated July 14, 2010 (ECF No. 9).

After Dr. Harum agreed to provide an expert report, petitioner made numerous filings supporting a request for advance payment of her fees. On January 26, 2012, petitioner filed additional medical records. See Pet'r's Exs. 20a-20d.[9] On August 27, 2013, a decision was entered denying petitioner's request for interim advanced costs for expert fees. Decision dated Aug. 27, 2013 (ECF No. 46).

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[8] The OAP was created to manage more than 5,400 petitions alleging that autism or autism spectrum disorder ("ASD") was caused by either the measles, mumps, and rubella ("MMR") vaccine or thimerosal, an ethylmercury preservative used in multi-dose vials of vaccines. See Autism General Order #1, dated July 3, 2002 (found at 2002 WL 31696785, 2002 U.S. Claims LEXIS 365; also available at http://www.uscfc.uscourts.gov/sites/default/files/autism/Autism+General+Order1.pdf (last visited on February 16, 2016)). Three special masters conducted separate proceedings in test cases involving the two theories of autism causation mentioned above. All found petitioners had not provided preponderant evidence of causation, indicating the cases were "not a close case." King v. Sec'y of Health & Human Servs., No. 03-584V, 2010 WL 892296, at *90 (Fed. Cl. Spec. Mstr. Mar. 12, 2010) (emphasis removed).

All three decisions were affirmed on appeal. Two of the three decisions in the Theory 1 test cases were appealed to the Federal Circuit. Cedillo v. Sec'y of Health & Human Servs., No. 98-916v, 2009 WL 331968 (Fed. Cl. Spec. Mstr. Feb. 12, 2009), aff'd 89 Fed. Cl. 158 (2009), aff'd, 617 F.3d 1328 (Fed. Cir. 2010); Hazelhurst v. Sec'y of Health & Human Servs., No. 03-654v, 2009 WL 332306 (Fed. Cl. Spec. Mstr. Feb. 12, 2009), aff'd, 88 Fed. Cl. 473 (2009), aff'd, 604 F.3d 1343 (Fed. Cir. 2010). Petitioners in the third test case did not appeal the Court of Federal Claims' decision. Snyder v. Sec'y of Health & Human Servs., No. 01-162v, 2009 WL 332044 (Fed. Cl. Spec. Mstr. Feb. 12, 2009), aff'd, 88 Fed. Cl. 706 (2009). Petitioners did not seek review of the special masters' decisions in the Theory 2 test cases. Dwyer v. Sec'y of Health & Human Servs., No. 02-1202v, 2010 WL 892250 (Fed. Cl. Spec. Mstr. Mar. 12, 2010); King v. Sec'y of Health & Human Servs., No. 03-584v, 2010 WL 892296 (Fed. Cl. Spec. Mstr. March 12, 2010); Mead v. Sec'y of Health & Human Servs., No. 03-215v, 2010 WL 892248 (Fed. Cl. Spec. Mstr. March 12, 2010). For further information and a comprehensive discussion of the OAP and proceedings after the conclusion of the test case litigation, see Sturdivant v. Sec'y of Health & Human Servs., No. 07-788, 2016 WL 552529 (Fed. Cl. Spec. Mstr. Jan 21, 2016).

[9] Although there is a later filed exhibit also labeled as exhibit 20, the undersigned will continue to use the exhibit numbers 20a-20d for the medical records filed on January 26, 2012, and exhibit number 20 for the later filed record. See Pet'r's Ex. 20 filed Sept. 23, 2014 (ECF No. 64).

Petitioner obtained counsel on April 17, 2014, and has since been represented by Mr. Peter Sarda. On September 23, 2014, petitioner filed Dr. Harum's expert report, curriculum vitae ("CV"), and medical literature. See Pet'r's Exs. 19-25 (ECF Nos. 63-64). Respondent filed a responsive expert report, CV, and medical literature from Dr. Gregory Holmes on December 17, 2014. See Resp's Exs. B-P (ECF No. 66). After Special Master Vowell raised specific criticisms of Dr. Harum's initial expert report,[10] petitioner filed the first supplemental report from Dr. Harum on March 9, 2015. See Pet'r's Ex. 26 (ECF No. 73). Petitioner also filed six of the references cited by Dr. Harum as one exhibit.[11] The case was reassigned to the undersigned on February 5, 2015.

On March 26, 2015, the undersigned held a status conference to discuss the current status of the case. Petitioner's counsel stated that he had been counsel of record in this case for less than one year. Although he admitted that Dr. Harum's report had, in certain parts, characterized this as an autism case, he reiterated petitioner's early arguments that her theory of causation involves encephalopathy as the injury, not autism. Order dated Mar. 30, 2015, at 1 (ECF No. 76). Counsel for respondent stated that as long as petitioner's expert characterized the child's injury as one that falls on the autism spectrum, the case could not be informally resolved. Id.

The undersigned then inquired about the status of testing results and medical records from the University of North Carolina ("UNC"), where B.A.B. was evaluated and underwent genetic testing. Order dated Mar. 30, 2015, at 1 (ECF No. 76). Specifically, when B.A.B. was seen at UNC, a report from Dr. Doreswamy questioned whether he had developmental impairment associated with a CIAS-1 mutation, with an incomplete clinical expression of neonatal onset multisystem inflammatory disease ("NOMID").[12] See Pet'r's Ex. 12 at 8-9. Dr. Doreswamy wrote, "Since the range of clinical CIAS-1 expression is broad, I will likely

[10] See Order dated Jan. 9, 2015 (ECF No. 68).

[11] See Amended Notice dated Mar. 10, 2015 (ECF No. 75) (providing the list of articles comprising Pet'r's Ex. 27 filed by CD on Mar. 11, 2015). Two of these articles were incomplete, containing only the first few pages, but petitioner re-filed the complete articles approximately three months later as Exhibit 28. See Notice dated June 8, 2015 (ECF No. 83) (providing the list of articles comprising Pet'r's Ex. 28, which was filed by CD on June 12, 2015.) It should be noted that petitioner filed multiple articles as one exhibit in both exhibits 27 and 28. The undersigned thus refers to all of these articles with the exhibit number to which petitioner assigned them, and the ECF page numbers have been provided for ease of reference.

[12] NOMID is a "very rare" disorder, which "causes persistent inflammation and tissue damage primarily affecting the nervous system, skin, and joints." https://ghr.nlm.nih.gov/condition/neonatal-onset-multisystem-inflammatory-disease (last visited Mar. 11, 2016). Individuals with the disorder have "a skin rash that is usually present from birth" and joint and skeletal abnormalities. Id. They "often have headaches, seizures, and vomiting resulting from chronic meningitis, which is inflammation of the tissue that covers and protects the brain and spinal cord (meninges)." Id. They may experience "[r]ecurrent episodes of mild fever," "[i]ntellectual disabilities," "[h]earing and vision problems," and "progressive kidney damage." Id.

advocate obtaining genetic analysis." Id. at 8-9. Petitioner was ordered to advise whether the testing was done, and if so, she was ordered to obtain and file the results. Order dated Mar. 30, 2015 at 1 (ECF No. 76). Moreover, in Dr. Harum's medical records filed by respondent, there is a reference to a C677Y mutation. Resp's Ex. A at 102. Petitioner was asked to investigate this question and file any records related to this gene mutation. Order dated Mar. 30, 2015 at 1 (ECF No. 76).

Also during the status conference on March 26, 2015, the undersigned addressed the fundamental flaw in Dr. Harum's expert reports: the lack of a foundational basis for her opinions. Order dated Mar. 30, 2015, at 2-3 (ECF No. 76). Petitioner was asked to provide a supplemental report by Dr. Harum addressing the basis for her opinion that B.A.B. had encephalopathy and to provide references from the medical records to support her claims. A review of B.A.B.'s medical records showed that he was developing normally at his nine, 12 and 15 month well-child visits. The Order stated that if petitioner was pursuing a claim that the vaccines B.A.B. received at his 12 month visit either caused or significantly aggravated an underlying condition, then petitioner would need to provide evidence that B.A.B. suffered an injury in the appropriate time frame in relation to his 12 month vaccinations. Lastly, the undersigned stated that if Dr. Harum was unable to substantiate her expert report, petitioner should consider dismissing her claim. Id. at 3.

On April 27, 2015, petitioner filed a status report indicating that she would be filing a motion for judgment on the record but that she needed additional time to obtain and file Dr. Harum's supplemental report, which would address the issues raised in the March 30, 2015 Order. Pet'r's Status Report dated Apr. 27, 2015 (ECF No. 77). Petitioner's request for additional time was granted. Scheduling Order dated Apr. 28, 2015 (ECF No. 78). A similar request for additional time was made on May 27, 2015, and again that request was granted. Order dated May 28, 2015 (ECF No. 80).

On June 3, 2015, petitioner filed a second supplemental expert report by Dr. Harum. See Pet'r's Ex. 29[13] (ECF No. 81). She also filed a motion for judgment on the record. Motion for Judgment on the Administrative Record ("MJR") dated June 3, 2015 (ECF No. 82).[14] In the motion, petitioner stated, "Because the medical records tell the story of [B.A.B.] since his birth, and the professional opinions support their respective positions, . . . no benefit to the parties would accrue by conducting a trial." MJR at 1. A few days later, petitioner filed medical literature cited by Dr. Harum.[15]

[13] Petitioner did not provide an exhibit number for this filing. Because she designated medical literature filed five days later as exhibit 28 (the next available exhibit number), the undersigned designates Dr. Harum's second supplemental report as exhibit 29. This report was also filed along with petitioner's motion for ruling on the record, as Tab 3. See Motion for Judgment on the Administrative Record dated June 3, 2015 (ECF No. 82).

[14] Along with her motion, petitioner filed copies of the previously filed expert reports: three by petitioner's expert, Dr. Harum, and one by respondent's expert, Dr. Holmes, as attachments 1-4. See also Pet'r's Exs. 19, 26, 29; Resp's Ex. B.

[15] See Notice dated June 8, 2015 (providing the list of articles comprising Pet'r's Ex. 28 filed by

Respondent filed her response on June 17, 2015, asserting that "[t]he [p]etition should be dismissed." Resp's Response dated June 17, 2015, at 13 (ECF No. 84). Respondent argued that "[p]etitioner has failed to provide any credible evidence that B.A.B. suffered an encephalopathy as defined by the Act, or that his neurological symptoms were in any way caused by the Proquad vaccine." Id. In accordance with the undersigned's August 19, 2015 Order, respondent filed a supplemental expert report by Dr. Holmes and additional medical literature on October 2, 2015. See Resp's Exs. Q, R (ECF No. 86).

This case is now ripe for adjudication of petitioner's motion for a ruling on the record.

II. Summary of Relevant Medical Records and Affidavits

a. Summary of Medical Records

B.A.B. was born on April 17, 2006. Pet'r's Ex. 2 at 1. He had a normal physical examination and his APGAR scores[16] were nine and nine. Id. His newborn screening tests and hearing examination were normal, and he was discharged home on April 19, 2006. Id. at 1-4. On May 20, 2006, petitioner called B.A.B.'s pediatrician complaining that B.A.B. had been crying for twelve hours, he was wheezing, and he seemed to be in pain. Pet'r's Ex. 4 at 27-28. On June 16, 2006, B.A.B. was noted to be spitting up and fretful. He was diagnosed with possible gastroesophageal reflux disease ("GERD") and mild reflux esophagitis. Pet'r's Ex. 6 at 1. The treating physician ordered Prevacid. Id. at 1-2. On June 23, 2006, at his two month well-child visit, he was noted to have nasal congestion. Pet'r's Ex. 6 at 11. During this visit, B.A.B. received the DTap, IPV, Hib, Prevnar, and Hepatitis B vaccinations. Pet'r's Ex. 3 at 2-3. No adverse reaction was noted. At his four month well-child visit on August 22, 2006, B.A.B. met all his developmental milestones. Pet'r's Ex. 6 at 12. On September, 21, 2006, he was diagnosed with an upper respiratory infection. Pet'r's Ex. 6 at 2-3.

At his six month well-child visit on October 25, 2006, B.A.B. again met all his developmental milestones. Pet'r's Ex. 6 at 13. B.A.B. received his DTap, IPV, Hib, Prevnar, and Hepatitis B vaccinations. Pet'r's Ex. 3 at 2-3. No adverse reactions were documented. On December 31, 2006, at about eight months old, B.A.B. had a fever of 102.5 but showed no other signs of serious infection. Pet'r's Ex. 4 at 22-26.

On January 1, 2007, B.A.B. presented to the emergency department ("ED") of Central Carolina Hospital ("CCH") with a fever of 105.9, decreased activity, excessive crying,

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CD on June 12, 2015). Petitioner re-filed five of the articles previously filed as part of exhibit 27, this time filing the complete articles for the two partially filed on March 11, 2015. Petitioner also included eight additional references, filing only the abstract or summary for three of these articles. See Pet'r's Ex. 28 at 48, 134-36.

[16] Appearance, Pulse, Grimace, Activity, and Respiration ("APGAR") score is a method of evaluating newborns to determine their overall health. See NELSON TEXTBOOK OF PEDIATRICS (19th ed. 2011) at 536-37.

congestion, and nasal discharge. Pet'r's Ex. 5 at 7-8. His oxygen saturation was excellent at 100 percent, he was otherwise well-appearing, he did not have a rash, and no speech or neurological problems were noted. Id. B.A.B.'s treating physicians diagnosed him with a fever, likely due to a virus. Pet'r's Ex. 6 at 2. While B.A.B. was in the ED, his father called the pediatrician's after hours telephone line, "agitated [and] demanding [that a] doctor meet [B.A.B.] in [the emergency room] as they do not know any of the doctors." Pet'r's Ex. 4 at 19. B.A.B.'s father further reported that his son had a "fever of 107." Id. Ultimately, B.A.B. left the ED against medical advice ("AMA"), and his father reported that he was taking his son to the pediatrician's office. Pet'r's Ex. 5 at 6. B.A.B. was seen on January 2, 2007, by his pediatrician, Dr. A.M. Hess. Dr. Hess documented that B.A.B. had fever up to 106, with minimal runny nose and cough. Pet'r's Ex. 6 at 2. His impression was that B.A.B. was experiencing a "fever, [which was] likely viral." Id. Dr. Hess saw B.A.B. the next day, January 3, 2007. Although he had experienced a fever for four days, B.A.B. appeared well during the visit and was in no distress. He had an ulcer on his tongue and one papule on his wrist, but otherwise, no rash was noted. His hemoglobin level was normal, and his complete blood count ("CBC") was "reassuringly viral." Id.

At B.A.B.'s nine month well-child visit on January 23, 2007, he again met all his developmental milestones, and his developmental assessment was normal. Pet'r's Ex. 6 at 14. He was using "lots of words" and was able to name colors. Id. On February 5, 2007, he had chapped cheeks and was diagnosed and treated for facial eczema. Id. at 3. On April 18, 2007, B.A.B. presented for his 12 month well-child visit and again met all of his developmental milestones. Id. at 15. His developmental assessment was normal. Id. He received the Prevnar, MMR, and Varicella vaccines,[17] and his physician did not note any adverse reaction. Pet'r's Ex. 3 at 2-3. There are no entries in the medical record that indicate B.A.B. had any abnormal neurological condition or symptoms following his 12 month vaccinations.

On July 13, 2007, B.A.B. presented to CCH for treatment of a lip laceration. Pet'r's Ex. 5 at 1. His past medical history was noted to be negative, and he appeared alert upon physical examination. Id. Neurological examination documented that he was oriented, that his mood and affect was normal, and that he had no sensory or motor deficit. Id. at 2. The nursing record from that admission notes that his mental status was alert and oriented and that his behavior was cooperative. Id. at 4. B.A.B.'s parents made several follow up calls to the pediatrician's office about the laceration on his face, but no pre-existing conditions or evidence of any problems, other than the concern about the small laceration on his face, were noted. See Pet'r's Ex. 4 at 16; Pet'r's Ex. 6 at 3. During this time, there is no indication in the medical records that B.A.B. experienced any neurological problems, especially not any signs or symptoms of encephalopathy or encephalitis.

B.A.B. was 15 months old on July 17, 2007. The record does not indicate that he experienced any medical problems at this time. On August 15, 2007, at about 16 months of age, B.A.B. had a fever of 103.8, but he had no other symptoms. Pet'r's Ex. 6 at 3. He looked well hydrated and was not in acute distress. Other than some pharyngitis and nasal congestion, he appeared normal and had no other symptoms. Id. A month later, on September 15, 2007, B.A.B. had a fever with congestion. Pet'r's Ex. 4 at 13-15; Pet'r's Ex. 6 at 4. His doctor observed on September 17, 2007, that B.A.B. was "cranky" but easily consoled, and he appeared well.

[17] These are the vaccinations at issue.

Pet'r's Ex. 6 at 4. His tympanic membranes were red, but there was no pus or bulging, and he was diagnosed with a viral syndrome. Id. During these visits, there were no symptoms to suggest that B.A.B. had encephalopathy or encephalitis, nor is there anything in the record to suggest that the pediatrician even considered either of these diagnoses.

B.A.B. had his 18 month well-child visit on October 19, 2007. At that visit, petitioner expressed concerns about B.A.B.'s speech development. She stated that he still had the "same speech" and "level of skills" that he had at 12 months of age, and that while he was saying quite a few words, he was not saying new words. He did not seem interested in acquiring new words, did not respond to his name, but did seem to hear normally. Pet'r's Ex. 6 at 4. B.A.B. did meet all of his developmental milestones. Id. at 16. He was drinking from a sippy cup, was able to say three words plus "mama" and "daddy," and was able to walk, run and climb. He was able to scribble, play games, and he tried to use a spoon and fork. Id. His gross motor development was normal. The pediatrician and petitioner decided to monitor B.A.B.'s developmental progress. The pediatrician reported, "mom will call [on the] first of the year if [B.A.B.] seems to have progressed no further." Id. at 4. During this visit there was no mention of or suggestion that B.A.B. had encephalopathy or encephalitis, or that he had any prior history of either of these conditions.

Approximately one month later, on November 23, 2007, B.A.B. presented to his pediatrician with fever, lack of appetite, and lethargy. He was diagnosed with right otitis media, and antibiotics were prescribed. Pet'r's Ex. 6 at 4. During that visit, the pediatrician noted that B.A.B. did not speak during the examination, that he did not point, and that he does not "bring mom into his world if he sees something." The pediatrician questioned "possible Autism Spectrum Disorder." Id. Later that same day, however, when B.A.B. was seen for a possible allergic reaction to amoxicillin, he was noted to be "doing well" and was running around the clinic, looking at pictures, and pointing to baby pictures on the walls. Id. at 5. In a follow-up visit on November 29, 2007, B.AB.'s pediatrician noted that he appeared healthy, playful, and was eating and drinking normally. Id. at 5-6. The pediatrician further observed that B.A.B. "seems to exhibit age appropriate behavior, maybe a litlte [sic] bit on the hyperactive side," and that "he does not exhibit any signs of systemic illness." Id. However, his doctor reported that petitioner was "clearly very preoccupied with what she perceives to be [B.A.B.'s] behavioral disorder." Id. at 5. The doctor further recommended "let[ting] the evaluation from TEACCH[18] occur first before we make any pronouncement as to whether B.A.B. does have any behavior abnormalities." Id. B.A.B.'s pediatrician noted that a lot of petitioner's concerns "have been prompted by her reading … on the internet." Id. at 6. The pediatrician recommended that petitioner "stick to only resources recommended to her by TEACCH or other reliable healthcare professionals." During these visits, there was no reference to any prior history of encephalopathy or encephalitis, or any prior or current diagnosis of either condition.

[18] Treatment and Education of Autistic and Related Communication Handicapped Children ("TEACCH") is a program that provides training and services to families with autistic children. The program was developed by the University of North Carolina at Chapel Hill. See UNC School of Medicine, "TEACCH Autism Program," available at https://www.teacch.com/ (last visited April 4, 2016).

On December 6, 2007, a developmental evaluation was performed by Donna A. Merkwan, Ph.D., an Infant-Toddler-Family Specialist ("ITFS"). The report was issued on January 22, 2008. Pet'r's Ex. 7 at 1-3. B.A.B. was referred for testing by his father due to concerns over his speech development and possible autism. Id. at 2. Entry level tests and results were as follows: gross and fine motor skills were within normal limits, cognitive skills were within normal limits, there was a 58 percent delay for chronological age for total language, self-help/adaptive behavior was within normal limits, and there was a 37 percent delay for chronological age for socio-emotional Early Learning Accomplishment Profile ("E-LAP"). Id. Dr. Merkwan questioned whether B.A.B.'s delay in social skills might be "related to his decreased interaction with other persons." Id. at 3. Dr. Merkwan also noted that while some of his repetitive behaviors may seem "autistic-like," they did not "constitute a definitive diagnosis for Autism Spectrum Disorder but may [] relate[] to [a] sensory processing disorder or to sensory integration issues." Id. Dr. Merkwan recommended that B.A.B. undergo evaluation by the Early Intervention Service Coordinator ("EISC") to determine his eligibility for services through the state's Infant Toddler Program. Id. Dr. Merkwan did not note any association between B.A.B.'s vaccines and his delay in language or socio-emotional skills. Dr. Merkwan did not note any prior history consistent with encephalopathy or encephalitis, and she did not consider any such prior or current diagnosis.

On January 17, 2008, Dana Miller, a speech therapist, evaluated B.A.B. Ms. Miller noted that a Receptive-Expressive Emergent Language Test, Third Edition (REEL-3) revealed that B.A.B. had a 38 percent language delay, and she recommended speech therapy twice weekly. Pet'r's Ex. 10 at 3-4. Occupational testing was performed January 18, 2008, by Melanie Lee, M.S. She administered the Peabody Developmental Motor Scales, Second Edition ("PDMS-2") test to measure B.A.B.'s developmental skills. Based on the test, B.A.B. was age appropriate in "visual-motor integration and grasping skills." Id. at 6. However, based upon B.A.B.'s mother's answers to the Infant/Toddler Sensory Profile Caregiver Questionnaire, B.A.B. experienced "differences in how he processes some types of sensory information." Id. Ms. Lee recommended occupational therapy ("OT") sessions once weekly. Id. at 8.

Additional evaluations were conducted in January and February 2008. On February 4, 2008, B.A.B. underwent a psychological evaluation by John Wilson, MA, LPA, using the Bayley Scales of Infant Development, Third Edition ("BSID-III"), Cognitive Scale test. Pet'r's Ex. 7 at 7. During the evaluation process, Mr. Wilson noted that B.A.B.'s family was concerned that his prior episode of elevated temperature of 107.5 may have impacted his development. B.A.B.'s score was 100, which "falls in the classification of average performance." However, he was performing at approximately a 21 month age equivalence. Id. B.A.B. was also evaluated using the Childhood Autism Rating Scale ("CARS") screening, a tool used to identify autism in children. B.A.B.'s score of 30 was "squarely on the line between non-autistic and the lowest bound of the mildly/moderately autistic range." Id. B.A.B.'s scores were inconsistent with a diagnosis of autism but placed him "on the threshold of symptoms of a mild/moderate autism spectrum disorder." Id. at 8.[19] It was recommended that B.A.B. see a pediatric neurologist and that he be evaluated for the TEACCH program. Id. Mr. Wilson did not note any prior history

[19] The International Statistical Classification of Diseases ("ICD") billing code for this visit to Dr. Harum was 315.5, which represents Mixed Development Disorder. Pet'r's Ex. 7 at 8.

consistent with encephalopathy or encephalitis, nor did he note any such prior or current diagnosis.

On September 26, 2008, B.A.B. underwent an evaluation by Dr. Karen Harum. Resp's Ex. A at 115. Dr. Harum tested B.A.B. for metabolic and neurological disorders in 2009 and 2010. Pet'r's Ex. 19 at 1. Genetic testing completed on October 16, 2008, for methylenetetrahydrofolate reductase ("MTHFR") mutations C677T and A1298C, revealed that B.A.B. had a single copy of the C677 mutation. Resp's Ex. A at 102-03. Genetic counseling was recommended. Id. at 103. Subsequent testing ordered by Dr. Harum reviewed that on March 4, 2009, B.A.B. had Rubeola AB (Antibodies), IgG, EIA, of 4.79H. Reference limits stated that greater than 1.09 was positive. Id. at 72. Lab results noted that "[p]resence of antibodies to Rubeola is presumptive evidence of immunity except when active infection is suspected." Id. Additional tests for complete blood count with differential and comprehensive metabolic panel returned all normal results, except for elevated blood urea nitrogen ("BUN")/Creatinine Ratio of 59 H, with a reference range of eight to 27. Id. at 71. Testing for interleukin-2 receptor alpha ("IL-2R") performed in September 2008 and March 2009 showed that B.A.B.'s levels were elevated. Id. at 72, 103. B.A.B.'s Neuron-specific enolase was elevated in September 2008, but repeat testing showed that this was normal. Id. at 103, 72. Repeat testing of B.A.B.'s BUN/creatinine ratio remained elevated with a score of 52 in March 2010, and a repeat test of IL-2R alpha performed in March 2010 was normal. Id. at 70. Vitamin D and Ferritin testing completed in March 2010 revealed low results. Id. On October 8, 2009, B.A.B. had an EEG, which was normal. Id. at 94.

On October 7, 2008, at B.A.B.'s two year old well-child visit to his pediatrician,[20] he met all of his developmental milestones. Pet'r's Ex. 6 at 19. He was using two words together, drawing/scribbling, running, climbing, throwing a ball and kicking. His parents reported that B.A.B. was making progress in PT, OT, and speech therapy and that he was undergoing an evaluation for ASD. Id.

On April 14, 2009, when B.A.B. was almost three years old, he was seen by Dr. Vinod Doreswamy at UNC Hospital, located in Chapel Hill, North Carolina, due to his parents' concerns regarding his elevated measles titer, his underlying developmental delay, and his recurrent fevers. Pet'r's Ex. 12 at 6. With regard to B.A.B.'s positive measles titer, Dr. Doreswamy opined that this "does not seem to be reflective of anything else other than a good vaccine response." Id. at 8. Dr. Doreswamy also noted that B.A.B. was "playing appropriately with toys, had short meaningful sentences in response to question[s], and made good eye contact." Id. With regard to the recurrent fevers, Dr. Doreswamy recommended genetic testing and conservative measures, including antipyretics for fever. Id. Dr. Doreswamy did not diagnose B.A.B. with encephalopathy or encephalitis, nor did he note any prior history of either condition. Dr. Doreswamy did not implicate B.A.B.'s vaccinations as causally associated or related to any of his medical problems or his parents' concerns.

[20] The undersigned notes that B.A.B. was actually two and a half years old at this visit. See Pet'r's Ex. 6 at 19.

On July 6, 2009, B.A.B. was evaluated by Dr. Lynn M. Wegner of UNC to consult regarding further interventions for his developmental delay. B.A.B. was transitioned out of the North Carolina Infant Toddler Program when he reached three years of age. Pet'r's Ex. 12 at 1. Dr. Wegner reviewed B.A.B.'s prior testing results and progress with speech and OT therapies. Id. at 1-2. Dr. Wegner opined that B.A.B. had "made incredible progress with the therapies offered" and expressed her concern about B.A.B. being withdrawn from school system support. Id. at 4. Dr. Wegner recommended that B.A.B. undergo updated language and fine motor integration assessments, that his school reinstate his Americans with Disabilities Act ("ADA") therapy, and that he have an Individualized Education Program ("IEP") to address language issues and fine and gross motor issues. Id. Dr. Wegner did not diagnose B.A.B. with encephalopathy or note any prior history of encephalopathy or encephalitis. Dr. Wegner did not document any issues associated with B.A.B.'s vaccinations, nor did she note any association between B.A.B.'s vaccinations and his developmental delay.

On January 4, 2010, B.A.B. was given a diagnosis of autism (ICD 299.00).[21] See Pet'r's Ex. 20a at 19. On March 3, 2010, B.A.B. met the criteria to be placed on a waiting list for specialized services under the Community Alternative Program for Persons with Mental Retardation and/or Developmental Disabilities ("CAP-MR/DD"). Pet'r's Ex. 11 at 1. On April 5, 2010, at approximately four years of age, B.A.B. had swollen tonsils and congestion, and he was diagnosed with strep pharyngitis. Pet'r's Ex. 6 at 9. At that visit, the pediatrician noted that B.A.B. had features consistent with autism. Id.

B.A.B. underwent a psychological evaluation by Christine Hook, Ph.D., on June 14, 2010, at the age of four years and two months. Pet'r's Ex. 20a at 3. Dr. Hook administered a battery of tests, including the Wechsler Preschool and Primary Scale of Intelligence ("WPPSI-III"), Vineland Adaptive Behavior Scales ("Vineland-II"), Behavior Assessment System for Children ("BASC-2"), and Gillian Autism Rating Scale ("GARS-2"). In summarizing the test results, Dr. Hook noted that B.A.B. possessed good vocabulary skills and that his social skills had improved, but that he continued to struggle with following directions. Id. at 7. She also

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[21] At that time, ICD 299.00 was the diagnostic code for autism. Centers for Medicare & Medicaid Services, "ICD-9 Code Lookup," available at https://www.cms.gov/medicare-coverage-database/staticpages/icd-9-code-lookup.aspx?KeyWord=299.00&bc=AAAAAAAAAAAEAA%3d%3d&# (last visited April 4, 2016). Autism is characterized by "[p]ersistent deficits in social communication and social interaction across multiple contexts," as well as "restricted, repetitive patterns of behavior, interests, or activities." The Diagnostic and Statistical Manual of Mental Disorders (American Psychiatric Association, 4th ed. text revision 2000) ("DSM-IV-TR"). A comprehensive discussion of the symptoms of autism can be found at White v. Sec'y of Health & Human Servs., No. 04-337V, 2011 WL 6176064, at *4-9 (Fed. Cl. Spec. Mstr. Nov. 22, 2011). The DSM-IV-TR has since been replaced by the Diagnostic and Statistical Manual of Mental Disorders, (American Psychiatric Association, 5th ed. 2013) ("DSM-V"), which uses the term "ASD", but the discussion of the symptoms of autism found in White is still relevant. In the DSM-V, the symptoms recognized by the medical community at large as those of an ASD have not changed, but the criteria for diagnosis have been refined, and the distinctions drawn in the DSM-IV among the diagnoses of autistic disorder, PDD-NOS, and Asperger's disorder have been eliminated.

noted that he had a "high level of over activity and impulsivity" as well as issues with sensory processing. Id. Dr. Hook concluded that B.A.B. met the criteria for Autistic Disorder and recommended a number of treatment strategies, including OT and continued programs in the school setting. Id. at 7-9. During this evaluation, Dr. Hook noted that B.A.B.'s "mother expressed concerns that [B.A.B.'s] regression was . . . possibly [related] to vaccines he received." The physician records indicate that the improbability of this correlation was discussed with B.A.B.'s parents. Id. at 3.

b. Statements from B.A.B.'s Parents

The record includes statements given by B.A.B.'s father, Tre Benson, and his mother, petitioner Erica Benson (Fester). See Pet'r's Ex. 14; Pet'r's Ex. 15. Mr. Benson stated that shortly after B.A.B.'s first birthday and shortly after receiving the MMRV vaccine, his cousin, who was approximately B.A.B.'s same age, came over to stay at B.A.B.'s home for about one week. During this time, Mr. Benson stated that B.A.B.'s cousin was often fussy, which made B.A.B. very angry. He reported that "[B.A.B.] smacked his cousin every chance he got" and would have "frequent temper tantrums," some of which would last for over an hour. Pet'r's Ex. 14 at 1. Mr. Benson reported that shortly thereafter, B.A.B. bit petitioner and that his temper tantrums worsened to the point that B.A.B. would not look his parents in the eye. Id. at 2.

Mr. Benson also reported events from the day that B.A.B. received the MMRV vaccination on April 18, 2007. He stated that B.A.B. had been crying and upset all day after receiving the vaccines and that afterwards he stopped talking altogether and did not say anything for almost a year. Pet'r's Ex. 14 at 2-3. Mr. Benson stated, "[t]hings sort of started rolling downhill from there, little by [little, my] son started eroding into a different little boy." Id. at 3. Mr. Benson detailed the progression of his son's doctor's appointments leading to his diagnosis of autism, highlighting the difficult impact of his son's disease on the family. Id. at 6.

Petitioner's statements similarly highlighted the alleged changes in her son's behavior that seemed to arise shortly after his vaccinations on April 18, 2007. She reported that B.A.B. "wouldn't stop crying or being upset, had a fever[,] and wouldn't take his normal nap" that afternoon. Pet'r's Ex. 15 at 1. Petitioner stated that after April 2007, B.A.B. exhibited hostile behaviors, he became interested with repetitive actions, and his eye contact lessened. She further reported that "he was always sick with a cold or mild illness and always had a low fever." Id. She described that as her son's condition worsened, he "stopped responding to his name or looking at us, not noticing or caring if we were in the room." Id. Given her growing concern, she eventually sought child development services to evaluate her son. Id.

III. Standards for Adjudication

The Vaccine Act established the Program to compensate vaccine-related injuries and deaths. § 10(a). "Congress designed the Vaccine Program to supplement the state law civil tort system as a simple, fair and expeditious means for compensating vaccine-related injured persons. The Program was established to award 'vaccine-injured persons quickly, easily, and with certainty and generosity.'" Rooks v. Sec'y of Health & Human Servs. 35 Fed. Cl. 1, 7 (1996) (quoting H.R. REP. NO. 908 at 3, reprinted in 1986 U.S.C.C.A.N. at 6287, 6344).

To receive compensation under the Program, petitioner must prove either: (1) that B.A.B. suffered a "Table Injury"—i.e., an injury listed on the Vaccine Injury Table—corresponding to a vaccine that he received, or (2) that B.A.B. suffered an injury that was actually caused by the vaccine (or vaccines) he received. See §§ 13(a)(1)(A) and 11(c)(1); Capizzano v. Sec'y of Health & Human Servs., 440 F.3d 1317, 1319-20 (Fed. Cir. 2006).

Although the Vaccine Table includes an injury of encephalopathy (or encephalitis) suffered five to 15 days after administration of the MMRV vaccine or one of its components,[22] petitioner is not alleging a Table Injury. See Petition at 1. Moreover, in order to qualify as a Table Injury, the encephalopathy petitioner claims B.A.B. suffered would have to satisfy the more narrow definition of encephalopathy contained in the Qualifications and Aids to Interpretation ("QAI") section of the Vaccine Injury Table.[23] The medical records and expert reports from Dr. Harum would not support an allegation of a Table encephalopathy, even if made.

Because petitioner cannot show that B.A.B. suffered a Table injury, she must prove that a vaccine B.A.B. received caused his injury. To do so, she must establish, by preponderant evidence: (1) a medical theory causally connecting a vaccine and B.A.B.'s injury ("Althen Prong One"); (2) a logical sequence of cause and effect showing that a vaccine was the reason for his injury ("Althen Prong Two"); and (3) a showing of a proximate temporal relationship between a vaccine and his injury ("Althen Prong Three"). Althen v. Sec'y of Health & Human Servs., 418 F.3d 1274, 1278 (Fed. Cir. 2005); § 13(a)(1) (requiring proof by a preponderance of the evidence).

IV. Expert Opinions

In support of her claim, petitioner offers the expert reports of Dr. Harum. See Pet'r's Exs. 19, 26, 29. Respondent provides the expert reports of Dr. Holmes. See Resp't Exs. B, Q. Both experts disagree as to the nature and cause of B.A.B.'s condition. Furthermore, Dr. Holmes extensively criticizes the factual predicates upon which Dr. Harum bases her opinion.

[22] 42 C.F.R. § 100.3(a)(III)(B) (2015).

[23] See 42 C.F.R. § 100.3(b)(2). As explained in Waddell, "[t]he scope of the medical term 'encephalopathy' is more expansive than the narrower, statutory definition set forth in the Table." Waddell v. Sec'y of Health & Human Servs., No. 10-316V, 2012 WL 4829291, at *12 (Fed. Cl. Spec. Mstr. Sept. 19, 2012) (referencing Hazlehurst v. Sec'y of Health & Human Servs., No. 03-654V, 2009 WL 332306, at *26-29 (Fed. Cl. Spec. Mstr. Feb. 12, 2009)). Encephalopathy as generally used means "any degenerative disease of the brain." DORLAND'S ILLUSTRATED MEDICAL DICTIONARY, 614 (32nd ed. 2012) ("DORLAND'S"). "The QAI definition of acute encephalopathy simply does not encompass every type of brain dysfunction to which the broader meaning of 'encephalopathy' applies." Blake v. Sec'y of Health & Human Servs., No. 03-31V, 2014 WL 2769979, at *6 (Fed. Cl. Spec. Mstr. May 21, 2014).

a. Petitioner's Expert, Dr. Karen Horton Harum

Dr. Harum is a board-certified pediatrician and specializes in the area of neurodevelopmental disabilities. Pet'r's Ex. 19 at 6. She attended medical school at the University of Miami School of Medicine and completed her internship at the University of Florida School of Medicine, Shands Teaching Hospital. Dr. Harum completed her residency in pediatrics at the University of Miami School of Medicine. She then completed a fellowship in neurodevelopmental pediatrics at the Kennedy Krieger Institute, at the Johns Hopkins University School of Medicine. Thereafter, she served as a post-doctoral fellow at the Kennedy Krieger Research Institute and received a National Research Service Award in Neuroscience. Dr. Harum is currently in private practice in Wilmington, North Carolina, at the Clinic for Special Children. Id. at 4-8.

i. B.A.B.'s Injury and Diagnosis

The most comprehensive explanation of Dr. Harum's opinions in this case is set forth in her third expert report (second supplemental report). As in all of her other reports, Dr. Harum describes B.A.B.'s injury as "subclinical encephalopathy, the manifestations of which are developmental regression, loss of language and sensory processing deficits." Pet'r's Ex. 29 at 4, filed June 3, 2015 (ECF No. 81). Dr. Harum defines "subclinical" encephalopathy as a subtle but not acute process that the parents could recognize, but "not obvious enough for the pediatrician to be concerned about." Id. She defines "encephalopathy" as a "sub-acute neurologic dysfunction: language plateau, loss of language, loss of auditory awareness, loss of visual attention, and decreased level of activity." Id. at 5. More specifically, Dr. Harum defines "regressive encephalopathy" as "neurological impairment of unknown cause that is associated with a period of past or on-going regression." Id. She explains that "30 [percent] of children ultimately diagnosed with autism have experienced developmental regression and other neurological changes, including subtle EEG changes." Id.

In her initial expert report, Dr. Harum describes B.A.B. as suffering symptoms "indicative of encephalopathy," and adding that "the symptoms of associated with Autism Spectrum Disorder are nearly identical." Pet'r's Ex. 19 at 3. She expands on this description in her second expert report (first supplemental report), opining that based on her "assessment at 29 months of age and the DSM-IV criteria, [B.A.B.] met [the]3 criteria for high functioning autism following encephalopathic regression." Pet'r's Ex. 26 at 4. Dr. Harum states that her diagnosis is consistent with John Wilson, Ph.D.'s diagnosis of autism when B.A.B. was 21 months of age, and Dr. Lynn Wegener's diagnosis of autism at 39 months of age.

ii. Medical Theory of Causation

In her initial expert report, Dr. Harum opined that vaccines, including the measles vaccine, can cause infectious encephalitis, particularly in immune compromised children, and that encephalitis may thereafter result in developmental regression. Dr. Harum quotes the Institute of Medicine ("IOM")'s Adverse Effects of Vaccines: Evidence and Causality,[24] as follows:

[24] Institute of Medicine ("IOM"), Adverse Effects of Vaccines: Evidence and Causality, 110

> There is mechanistic evidence of vaccine reactions that does account for acute onset of infections and deterioration, but also for delayed onset of infection, delayed up to [nine] months after vaccine administration. For example, the measles vaccine can cause measles inclusion body encephalitis due to the vaccine strain of measles, after administration of the measles vaccine, particularly in immune compromised individuals up to [nine] months after the vaccine administration.

Pet'r's Ex. 19 at 2 (quoting IOM at 110).

Dr. Harum further posited that "there is a plausible body of evidence to associate MMRV and other vaccines and encephalitis resulting in mental retardation and speech impairment. Therefore, one can postulate that there is a plausible association between MMRV and other vaccines and subacute encephalopathy associated with developmental regression." Pet'r's Ex. 19 at 2.

Subsequently, former Chief Special Master Vowell identified a number of problems with Dr. Harum's medical theory as stated in her initial expert report. See Order dated Jan. 9, 2015 (ECF No. 68). First, "[t]he medical records and the parents' early statements do not support the existence of encephalitis after the 12 month vaccinations." Id. at 1-2. Additionally , "Dr. Harum mention[ed] "'subclinical' encephalitis [in her report] but fail[ed] to point to any evidence that one existed, or that subclinical encephalitis can result in an autism diagnosis." Id. at 2. Moreover, "none of the articles or excerpts filed provide[] support for . . . Dr. Harum's theory: that the 12 month vaccines caused encephalitis that manifested with autism-like symptoms." Id. In conclusion, Chief Special Master Vowell stated, "[i]n essence, Dr. Harum strings together reported effects from MMR vaccines to conclude that MMR can cause autism or cause a condition resulting in 'autism-like' symptoms, in spite of a dearth of evidence that B.A.B. had symptoms of encephalitis or encephalopathy in the months after his Proquad vaccination." Id.

In her next supplemental report, Dr. Harum conceded that "there [was] no evidence of encephalitis following the 12 month vaccinations." Pet'r's Ex. 26 at 1. After this concession, however, Dr. Harum changed tack. Instead of arguing that the 12 month vaccines caused an acute or delayed infectious encephalitis or encephalopathy, manifested with autism-like symptoms, in her second and third expert reports, Dr. Harum posited a multi-faceted framework, built on a series of presumptions. Dr. Harum opined that B.A.B. "contracted roseola infantum[25] at [nine] months of age, coincident with granulocytopenia and symptoms of encephalopathy, including language plateau." Pet'r's Ex. 26 at 3; accord. Pet'r's Ex. 29 at 8. Then, within "[three] months, while there was ongoing lymphocytosis and presumed brain inflammation, and in this immune dysregulated state, [B.A.B.] received a cluster of vaccines that contributed further to microglial activation, overactive Th2 immunity, and worsening encephalopathy." Pet'r's Ex.

(Kathleen Stratton et al. eds., 2012) (internal citations removed).

[25] Roseola Infantum is clinically defined as "a type of rose-colored rash seen most often in an infectious disease such as measles or other exanthematous diseases." DORLAND'S at 1654.

26 at 3. Dr. Harum argued that the vaccines further contributed to "this injury pathway through the load of aluminum, glutamate, formaldehyde and unidentified pathogens" found in the vaccines. Pet'r's Ex. 26 at 3; accord Pet'r's Ex. 29 at 8.[26] Dr. Harum writes that these mechanisms contributed to B.A.B.'s "encephalopathic regression through pathophysiologic mechanisms described repeatedly in neuronal injury in the scientific literature."[27] Pet'r's Ex. 29 at 9.

In her third and final supplemental report, Dr. Harum elaborates upon several direct questions posed by former Chief Special Master Vowell in an earlier scheduling order. See Order dated January 9, 2015 (ECF No. 68). Dr. Harum posits that B.A.B. had a C667T mutation, the significance of which was "very limited, except under circumstances of folate deficiency." Pet'r's Ex. 29 at 1. Dr. Harum then states, "[I]f other undiagnosed problems within the methylation cycle . . . are also present, it weakens the individual's ability to provide methyl groups to a wide variety of molecules. Under these circumstances, glutathione production can be limited by insufficient methylation cycle activity." Id. Dr. Harum continues by stating that issues with the methylation cycle can inhibit methyl production, which thus limits production of glutathione, which helps protect the body from toxins. Id. These inhibitions of methylation pathways, according to Dr. Harum, can cause "adjuvants such as alum [to] pose a greater toxic effect." Id. at 2.

Dr. Harum summarizes her opinion of how B.A.B. suffered an encephalopathy by first stating that B.A.B. suffered from roseola infantum in January 2007 at the age of nine months. She then states, "[roseola infantum] may have caused a mild and temporary encephalitis that predisposed [B.A.B.] to further neurological inflammation and insult." Pet'r's Ex. 29 at 3. However, in the same paragraph, Dr. Harum admits that B.A.B. "did not have seizures," that he "did not have [cerebro-spinal fluid] assays to document HHV-6 in the central nervous system," and that "he did not meet the criteria for encephalitis." Id. Surprisingly, however, Dr. Harum characterizes the alleged encephalopathy suffered by B.A.B. as "mild and temporary," which "predisposed him to further neurological inflammation and insult." Id. After he received his 12 month vaccines, Dr. Harum stated that B.A.B. then "suffered subclinical encephalopathy" between the age of 12 and 18 months, opining that his encephalopathy is "described as sub clinical because it was not acute and was subtle enough for parents to recognize, but not obvious enough for the pediatrician to be concerned about . . . ." Id. at 4.

[26] Here, Dr. Harum cited to Campbell et al., "Chronic Exposure to Aluminum in Drinking Water Increases Inflammatory Parameters Selectively in the Brain," 75 J. NEUROSCI. RES. 565-72 (2004) (complete article filed as Pet'r's Ex. 28 at 40-47); and M. Giangaspero et al., "Genotypes of Pestivirus RNA Detected in Live Virus Vaccines for Human Use," 63 J. VET. MED. SCI. 723-33 (2001) (abstract only, filed as Pet'r's Ex. 28 at 134).

[27] In support of this assertion, Dr. Harum cited Atladottir, Thorsen, Schendel, et al., "Association of Hospitalization for Infection in Childhood with Diagnosis of Autism Spectrum Disorders: A Danish Cohort Study," 164 ARCH PEDIATR. ADOLESC. MED., 470-77 (2010) (abstract only from the National Center for Biotechnology Information website, filed as Pet'r's Ex. 28 at 135-36).

Finally, Dr. Harum stated that several of B.A.B.'s laboratory test results indicate that he suffered some sort of subclinical encephalopathy. She points to abnormal folate insufficiency, oxidized DNA, reduced glutathione, irregular neuron specific enolase ("NSE") levels, abnormal IgG total count, and Il-2 receptor alpha elevation, positing that all of these "abnormalities" suggest that B.A.B. "clearly has an encephalopathy." Pet'r's Ex. 29 at 6.

To evaluate the viability of this theory, the undersigned will separately examine each of Dr. Harum's presumptions. The undersigned will review both the foundation for each presumption and the supporting medical literature. While the undersigned has reviewed and considered all the evidence in this case and the entire record as a whole, the following is by no means a complete recitation of all the relevant facts and evidence considered. See § 300aa-13(a) (stating that the special master should consider the "record as a whole").

1. Dr. Harum's Theory Part 1

The first presumption made by Dr. Harum is that B.A.B. contracted roseola infantum at nine months of age. Pet'r's Ex. 29 at 8. Dr. Harum initially saw B.A.B. when he was 29 months of age, so she necessarily relies on medical records for evidence of this part of her opinion. She writes that B.A.B. "contracted a febrile illness characterized by fever over 105 degrees at 9 months of age . . . later determined to be roseola infantum." Pet'r's Ex. 26 at 1.

The medical records, however, do not show that B.A.B. was diagnosed with roseola infantum at nine months of age. On January 1, 2007, B.A.B. presented to CCH ED with a fever of 105.9, decreased activity, excessive crying, congestion and nasal discharge. Pet'r's Ex. 5 at 7-8. B.A.B's oxygen saturation was excellent at 100 percent, he was otherwise well-appearing, he did not have a rash, and the treating physician did not observe any speech or neurological problems. Id. B.A.B. was diagnosed with a fever of likely viral origins. Pet'r's Ex. 6 at 2. On January 3, 2007, B.A.B. was again seen by his pediatrician, who noted that B.A.B. had an ongoing fever for four days but that he was "well appearing." Id. The pediatrician next saw B.A.B. on February 5, 2007, and at that time noted a rash but no fever. Id. at 3. B.A.B. was examined and evaluated by a pediatrician on three different dates during this febrile illness, but not once was he diagnosed with roseola infantum or noted to have any neurological abnormalities.

B.A.B. was not diagnosed with roseola infantum until February 4, 2008, at 22 months of age, when he presented at his pediatrician's office with a diffuse body rash, described as a "diffuse pink MP (maculopapular) rash which blanches on extremities and torso." Pet'r's Ex. 6 at 7. In addition to specifically describing the rash, the pediatrician documented that "this is a normal viral pattern specifically that of roseola." Id. At that point, B.A.B. was 22 months of age, and nearly 10 months had passed since he received the vaccines at issue. Thus, the undersigned finds Dr. Harum's opinion that B.A.B. suffered from roseola infantum at the age of nine months unpersuasive.

2. Dr. Harum's Theory Part 2

In order to continue the evaluation of Dr. Harum's theory, the undersigned next assumes, for the sake of argument, that B.A.B. did have roseola infantum at nine months of age, or that his

viral illness was the equivalent of that condition. Dr. Harum's next presumption is that the illness (i.e. the alleged roseola infantum) was "coincident with granulocytopenia and symptoms of encephalopathy including language plateau." Pet'r's Ex. 26 at 3. Granulocytopenia is defined as a "reduction in the number of granular leukocytes in the blood."[28] Dr. Harum opined, "Lab evidence demonstrated a . . . low granulocyte count." Id. at 1. B.A.B.'s blood work from January 3, 2007, does show decreased granular leukocytes. Id. at 25. B.A.B.'s pediatrician at the time documented that a CBC had been performed and that the results were "reassuringly viral." Id. at 2.

Thus, while Dr. Harum is correct that B.A.B. had granulocytopenia, in that he had decreased granular leukocytes, this was a product of a reassuring immune response to his viral illness, as evidenced by his pediatrician's contemporaneous record. Moreover, Dr. Harum failed to provide support for her position that roseola infantum or any other viral illness causes or contributes to encephalopathy or language plateau. And in fact, in her third supplemental report, Dr. Harum concedes that on April 18, 2007, "prior to [B.A.B.'s] cluster of vaccines, [his] granulocytopenia . . . resolved and he appeared quite healthy with normal developmental milestones." Pet'r's Ex. 29 at 3.

Despite her acknowledgment that B.A.B.'s roseola infantum resolved prior to receiving his 12 month vaccinations, Dr. Harum opined that roseola infantum is associated with HHV-6, "an encephalitic virus, with a predilection for brain infection and latent colonization of neuronal tissue." Pet'r's Ex. 26 at 1. However, she concedes that B.A.B. did not have seizures, that there was no testing of his cerebrospinal fluid, and thus that there is no evidence of HHV-6 in his brain. Nevertheless, she argues that this HHV-6 infection, of which there is no evidence, caused encephalopathy, "based on the history of regression." Id. B.A.B. did not show regression at nine months, and according to his pediatrician's records and his parents' early statements, he did not experience any developmental delays until he was 15 months old. There are no facts to support Dr. Harum's underlying premise that B.A.B. had any abnormal process in play prior to receiving his 12 month vaccinations. Thus, there is no basis for the first tenet of Dr. Harum's theory, that B.A.B. "contracted roseola infantum at 9 months of age, coincident with granulocytopenia and symptoms of encephalopathy including language plateau." Id. at 3.

Similarly, Dr. Harum argues that the vaccines were given in proximity to a "significant encephalitic virus infection." Pet'r's Ex. 26 at 3. In her later report, the "significant encephalitic virus infection" is downgraded to a "mild and temporary encephalitis." Pet'r's Ex. 29 at 3. Dr. Harum conceded, however, that B.A.B. "did not meet the criteria for encephalitis." Id. She specifically stated, "I concede that there is no evidence of encephalitis following the 12 month vaccinations." Id. at 4. Moreover, the medical literature Dr. Harum cites to support this part of her proposed medical theory is insufficient to show roseola, or any other viral illness, coincident with one transient episode of granulocytopenia, causes or is diagnostic of encephalitis or encephalopathy, manifest by language plateau, which "predisposed [B.A.B.] to further neurological inflammation and insult." Id.

Dr. Harum claims the "mechanisms of injury" she describes are "described repeatedly" in

[28] DORLAND'S at 803.

scientific literature and cites Atladottir, et al.[29] to support her claim. Pet'r's Ex. 29 at 9. However, petitioner has filed only the abstract of this article, and it does not provide the support Dr. Harum claims. See Pet'r's Ex. 28 at 135-36. The abstract describes a Danish population-based cohort study that examines whether there is an association between infections requiring hospitalization and ASDs. Id. at 135. The study found that there was an increase in ASD diagnoses and mental retardation in these hospitalized children, but the authors observed that this association was found for children suffering from noninfectious diseases and infectious diseases that were both bacterial and viral in origin. The authors specifically concluded that the association found did not suggest causality because the association was observed throughout these different groups. Id.

To support her claim that "mechanisms [of injury] converge onto the more recently described neuronal pathways and signaling systems identified as impaired in autism," Dr. Harum cites the Pinto article.[30] This article examines the genetic variations involved in ASDs. Pet'r's Ex. 27 at 35. The authors concluded that the studies performed "highlighted a striking degree of genetic heterogeneity, implicating both de novo germline mutation and rare inherited ASD variation distributed across numerous genes." Id. 36. It did not, however, provide support for Dr. Harum' claims. The article does not mention vaccines.

Repeatedly throughout her expert report, Dr. Harum makes conclusory statements that language plateau, developmental regression, and sensory processing deficits are symptoms or manifestations of encephalopathy. Pet'r's Exs. 26 at 1; 29 at 4. But she does not offer any support for these conclusory statements, especially in light of the facts and circumstances of this case. Moreover, these are some of the most common symptoms of ASD. See White, 2011 WL 6176064, at *4-9. B.A.B.'s first symptom, speech delay, is generally the first symptom observed by parents of autistic children. Id. at *8. Dr. Harum does not provide any evidence to support a conclusion that B.A.B.'s symptoms should be categorized as encephalopathy rather than autism.

Dr. Harum stated that because B.A.B. had an elevated neuron specific enolase ("NSE") level of 17.4 on September 29, 2008, approximately one and one-half years after his vaccinations, this is evidence of neuronal death and encephalopathy. Pet'r's Ex. 29 at 6; see Pet'r's Ex. 9 at 10 (reporting B.A.B.'s test results). She does not explain, however, why B.A.B. would have normal NSE results six months later, on March 4, 2009, if he were experiencing "ongoing neuronal injury." See Pet'r's Ex. 29 at 6.

In support of her opinion that B.A.B.'s elevated NSE levels are consistent with an "ongoing neuronal injury," Dr. Harum cited a study by Berger, et al.[31] regarding infants and

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[29] See supra note 27.

[30] Pinto et al., "Convergence of Genes and Cellular Pathways Dysregulated in Autism Spectrum Disorders," 94 AM. J. HUMAN GENETICS, 677-94 (2014) (filed twice at Pet'r's Ex. 27 at 35-52 and Pet'r's Ex. 28 at 86-103). The undersigned will cite to the article filed as part of Pet'r's Ex. 27.

[31] Berger et al., "Neuron-Specific Enolase and S100B in Cerebrospinal Fluid After Severe Traumatic Brain Injury in Infants and Children," 109 PEDIATRICS E31 (2002) (filed as Pet'r's Ex

children who have experienced traumatic brain injuries ("TBI"). See Pet'r's Ex. 27 at 2. The Berger researchers surveyed 10 pediatric patients ranging in ages from two months to nine years who were admitted to the Children's Hospital of Pittsburgh with both inflicted and non-inflicted[32] TBI. Id. at 3. The researchers tested the children's cerebrospinal fluid ("CSF") for the presence of NSE, hypothesizing that the concentration of NSE in children is higher after a TBI. Indeed, the study found that patients with inflicted TBI experienced "an initial peak in NSE concentration on day 1 after injury followed by a second, higher peak that was sustained for up to 8 days." Id. at 4. In patients with non-inflicted TBI, "the initial concentration [of NSE] was the peak concentration," meaning that the patients experienced their peak NSE levels shortly after the initial injury that lead to the TBI. Id.

Dr. Harum's citation to the Berger study in support of her contention that B.A.B. experienced an "ongoing neuronal injury" is erroneous for several reasons. First, petitioner does not contend, and the record does not reflect, that B.A.B. ever suffered from an inflicted TBI. However, even if B.A.B. had suffered an inflicted TBI, the NSE levels from the patients in the Berger study peaked one day after injury and were only sustained for up to eight days after injury. Pet'r's Ex. 27 at 4. B.A.B.'s elevated NSE levels were not measured until one and a half years after he received the MMRV vaccination at issue here. Thus, the results of the study are inapplicable to B.A.B. The Berger study actually disproves Dr. Harum's theory that B.A.B.'s elevated NSE levels point to an "ongoing neuronal injury" that resulted from the vaccinations he received on April 18, 2007. Assuming the Berger study is applicable to B.A.B.'s case, the results of the study suggest that his elevated NSE levels on September 29, 2008, were actually the result of a much more recent injury than the vaccines he received more than five months earlier.

3. Dr. Harum's Theory Part 3

The next part of Dr. Harum's medical theory is that within "3 months [of having roseola – at age nine months], while there was ongoing lymphocytosis[33] and presumed brain inflammation, and in this immune dysregulated state, [B.A.B.] received a cluster of vaccines that contributed further to microglial activation, overactive Th2 immunity, and worsening encephalopathy." Pet'r's Ex. 26 at 3.

With respect to Dr. Harum's contention that B.A.B. experienced an "ongoing lymphocytosis," a CBC with differential was performed on January 3, 2007, when B.A.B. had a fever and was diagnosed with a viral illness. See Pet'r's Ex. 6 at 25. B.A.B.'s lymphocyte levels

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27 at 1-8).

[32] The Berger researchers note that there are two sub-groups of TBI: inflicted and non-inflicted. Pet'r's Ex. 27 at 2. "The mechanism of [inflicted] TBI – violent shaking often followed by impact with a hard surface – is unlike any of the mechanism of [non-inflicted] TBI and is particularly deleterious to the brain." Id. at 3.

[33] Lymphocytosis is defined as an "excess of normal lymphocytes in the blood." DORLAND'S at 1085.

were normal at that time. Id. The CBC was repeated on April 18, 2007, at the time of his 12 month checkup when he received the vaccinations at issue in this case, and again his lymphocyte levels were in the normal range. Id. at 24. Dr. Harum writes that on April 18, 2007, B.A.B. had a "low normal granulocyte count and high lymphocyte count . . . reflecting ongoing activation following a viral infection." Id. at 2. But the laboratory results identify the white blood cell count, the granulocyte count, and the lymphocyte count as being within normal limits. Id. at 24.

Furthermore, there is no indication in the pediatrician's records that B.A.B. experienced any "ongoing activation" or other abnormal immune response. See Pet'r's Ex. 6 at 15. In fact, at his 12 month visit, B.A.B.'s physical exam, including his neurological exam, was completely normal. Id. No parental concerns are noted and no follow up was needed. Id. B.A.B.'s parents did not call or visit his pediatrician after the 12 month visit until July 13, 2007, when B.A.B. lacerated his lip. There is no indication of illness whatsoever during this time frame. Moreover, on October 7, 2008, at approximately 18 months of age, a repeat CBC again showed normal granulocyte and lymphocyte levels. Id. at 21. Dr. Harum's conclusions that B.A.B. had ongoing lymphocytosis or that he had "ongoing activation following viral infection" are erroneous. Pet'r's Ex. 26 at 2. There is simply no evidence that B.A.B. had any "immune dysregulation" following his viral illness that occurred in January 2007.

Next, Dr. Harum stated that B.A.B. had "presumed brain inflammation." Pet'r's Ex. 26 at 3. There is, however, no evidence of brain inflammation. The CBCs were reassuringly normal. See Pet'r's Exs. 6 at 21, 24, 25; 13 at 3.[34] Physical and neurological examinations were normal. Id. at 15. There is no foundation upon which Dr. Harum could base the presence of brain inflammation at any time leading up to B.A.B.'s 12 month vacations, or even after them.

Similarly, Dr. Harum used the phrases "neuronal inflammation," "neuronal irritation," and "neuronal injury" to describe B.A.B.'s health prior to receipt of his 12 month vaccinations. Pet'r's Exs. 26 at 3; 29 at 6. She stated that the vaccines were "delivered at a vulnerable period of immune dysregulation (discussed above) and neuronal[35] inflammation." Pet'r's Ex. 26 at 3. She also stated that the vaccines "independently elicit neuronal irritation, even doubling the risk of seizures." Id. Dr. Harum does not cite to any evidence in B.A.B.'s medical records, or provide any supporting literature to provide a basis for her statements that B.A.B. had either neuronal inflammation, seizures, or other injury. And as discussed above, the record lacks evidence of either symptom manifestation or diagnosis of such an injury either before or after B.A.B.'s 12 month vaccinations.

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[34] Blood tests (CBCs) were performed on October 7, 2008, April 18, 2007, January 3, 2007, and March 4, 2009, respectively. The results were mostly normal. The records do indicate decreased levels of WBCs and granulocytes on January 3, 2007, but these levels returned to normal and were normal on April 18, 2007, October 7, 2008, and March 4, 2009. Id.

[35] Neurons are classified as "any of the conducting cells of the nervous system. A typical neuron consists of a cell body, containing the nucleus and the surrounding cytoplasm; several short radiating processes; and one long process, which terminates in twiglike branches and may have branches projecting along its course. The axon together with its covering or sheath forms the nerve fiber." DORLAND'S at 1267.

The article cited by Dr. Harum to support her assertions regarding inflammation, Vargas, et al.[36], was discussed extensively in the OAP. The authors of that article "demonstrated a marked increase in neuroglial responses, characterized by activation of the microglial and astroglia in the brains of autistic patients." Pet'r's Ex. 27 at 24. They concluded that these increases were "likely part of neuroinflammatory reactions." Id.; see also Snyder, 2009 WL 332044 at, *87-88 (discussing Vargas). However, as the special masters in the OAP test case indicated, the authors did not discuss whether they believed the neuroinflammation found was a cause or effect of autism. See e.g., King, 2010 WL 892296 at, *40-41; Snyder, 2009 WL 332044, at *88. Moreover, there is no connection to or even the mention of a vaccine in the article.

The entire notion that BAB had some "identifiable predisposing conditions, such as metabolic abnormalities or immune deficiencies" which would cause him to be susceptible to increased microglial and astroglial activation from vaccines is wholly unsupported by the medical records. Pet'r's Ex. 29 at 5. Dr. Harum also concedes there is no evidence that B.A.B. had any relevant genetic abnormality that would have affected his development. Id. at 5-6.

4. Dr. Harum's Theory Part 4

The next part of Dr. Harum's medical theory is that B.A.B. "received a cluster of vaccines that contributed further to microglial activation, overactive Th2 immunity, and worsening encephalopathy." Pet'r's Ex. 26 at 3. She assumes that B.A.B. already had microglial activation, overactive Th2 immunity, and encephalopathy. Dr. Harum does not define microglial activation in her expert report, but she again references the article by Vargas, et al. to support her broad assertions.[37] However, in Vargas, the authors do not suggest that vaccines cause or contribute to any microglial activation they found.

Moreover, it is unclear what Dr. Harum means by referencing B.A.B.'s "overactive Th2 immunity," as a component of her theory. Presumably, it ties into the aspect of her theory related to the notion that B.A.B. had an abnormal immune response following his viral illness which was worsened by the vaccines and played a role in causing or contributing to encephalopathy. Throughout her reports, Dr. Harum suggests that B.A.B. may have an immune dysregulation, immune incompetence, or that he may have suffered an "untoward immunologic reaction." Pet'r's Ex. 26 at 3. She believes that B.A.B.'s abnormal granulocyte and lymphocyte counts are evidence of his immune incompetence. Id.

She also writes that B.A.B.'s "IgG total count was also high, suggesting excessive immunoglobulin mediated inflammation (Th2 predominance)." Pet'r's Exs. 26 at 2; 29 at 6. The IgG test to which Dr. Harum references, however, was performed on September 29, 2008, a year and a half after the vaccines were given. Dr. Harum provides no explanation for how that

[36] Vargas et al., "Neuroglial Activation and Neuroinflammation in the Brain of Patients with Autism," 57 ANN. NEUROL. 67-81 (2004) (filed as Pet'r's Exs. 27 at 16-30; 28 at 111-25).

[37] See Vargas et al., supra note 34.

result, 18 months later, provides evidence of immune mediated inflammation that contributed to autism or encephalopathy temporally associated with the vaccinations. She also cites an article by Asherman, et al.,[38] which examined the pro-inflammatory and anti-inflammatory characteristics of IgG. See Pet'r's Ex. 28 at 104-10. The authors devoted much of the discussion to the anti-inflammatory aspects of IgG, theorizing that a better understanding of this aspect "might enable us to replace this primary blood product [(IVIG)[39]] in the future with a recombinant therapeutic." Id. at 105.

One other test relied upon by Dr. Harum is the IL-2 receptor Alpha, which was found to be elevated on September 29, 2008, and March 4, 2009. Pet'r's Ex. 9 at 10; Pet'r's Ex. 13 at 1. Dr. Harum states that these results suggest "immune system activation." Pet'r's Ex. 26 at 2. Again, she does not explain how this fact supports her theory or provides evidence that B.A.B. suffered a vaccine-related injury.

Dr. Harum further cited an article by Triger, et al.[40] for the proposition that "highly elevated measles antibodies have been seen in a variety of clinical and autoimmune disorders, characterized by excessive antibody production and inadequate innate immunity." Pet'r's Ex. 26 at 3. Examining groups of 21 to 65[41] patients (depending on the disease involved), the authors sought to determine if the elevated measles antibody titers reported in patients with chronic active hepatitis could also be observed in patients with other diseases. Pet'r's Ex. 28 at 126. The authors found the same result in patients with other diseases, such as DLE,[42] but not diseases such as rheumatoid arthritis. Id. at 126, 131.[43] This article, however, offers no support for Dr. Harum's assertions in this case.

As stated earlier, B.A.B.'s elevated measles antibody titer following the MMRV vaccination was characterized by Dr. Doreswamy as nothing more than "a good vaccine response." Pet'r's Ex. 12 at 8. And even Dr. Harum seems to doubt her commitment to the point that B.A.B.'s response to the measles vaccine was somehow evidence of immune

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[38] Ascherman et al., "The Other Side of Immunoglobulin G: Suppressor of Inflammation," 160 CLINIC. EXP. IMMUN. 161-67 (2010) (filed as Pet'r's Ex. 28 at 104-10).

[39] "IVIG" stands for intravenous immunoglobulin. Neil M. Davis, MEDICAL ABBREVIATIONS, 15th Edition, at 178 (2011).

[40] Triger et al., "Measles Antibodies and Autoantibodies in Autoimmune Disorders," 24 CLIN. EXP. IMMUNOL. 407-14 (1976) (filed as Pet'r's Ex. 28 at 126-33).

[41] The total for the group of patients with rheumatoid arthritis is listed as 55 at one point and 65 at another. Compare Pet'r's Ex. 28 at 126 with id. at 128 (total listed in table).

[42] The authors of this article do not provide the disease labeled as DLE but it appears to stand for discoid lupus erythematosus, "a chronic form of cutaneous lupus erythematosus." DORLAND'S at 1079.

[43] The authors acknowledged their result with regard to rheumatoid arthritis differed from other studies but argued those studies were not comparable. Pet'r's Ex. 28 at 131.

incompetence when she says, "[w]hile . . . robust antibody production against the vaccine strain of measles is good, it may also contribute to ongoing inflammation of the brain . . . ." Pet'r's Ex. 26 at 3. The word "may" in this context is weak when compared with the balance of Dr. Harum's opinions. In her third report, Dr. Harum finally conceded that B.A.B.'s "antibody response to measles was normal." Pet'r's Ex. 29 at 7.

The last phase of this part of Dr. Harum's theory is that B.A.B. "received a cluster of vaccines that contributed further to . . . worsening encephalopathy." Pet'r's Ex. 26 at 3. The lack of evidence to support any conclusion that B.A.B. had encephalopathy has been previously discussed. Here, however, Dr. Harum implies that B.A.B.'s condition is worsening, and again, she fails to cite to any medical records to provide a basis for this characterization.

5. Dr. Harum's Theory Part 5

The last aspect of Dr. Harum's medical theory is that the vaccines further contributed to "this injury pathway through the load of aluminum, glutamate, formaldehyde and unidentified pathogens." [44] Pet'r's Exs. 26 at 3; 29 at 8. Dr. Harum stated that B.A.B. received "225 mcg in the PedVax and 125 mcg in [the] Proquad" vaccines, for "a total of 350 mcg aluminum." Pet'r's Ex. 29 at 8. Dr. Harum further stated that the "FDA recommends no more than 25 mcg of aluminum per day, when parenterally delivered." Id. However, Dr. Harum provided no citation for her assertion, nor did she discuss the potential effect that the alleged larger dose of aluminum in B.A.B.'s vaccinations could have had on his development.

Furthermore, Dr. Harum opined that in the context of the C677T MTHFR mutation, "the aluminum may have presented an additional aggravating factor." Pet'r's Ex. 29 at 8. She states generally that "vaccines can contribute further to this pathway of injury through the load of aluminum, glutamate, formaldehyde and unidentified pathogens." Id. She does not, however, explain how the aluminum may have been an aggravating factor, nor does she provide any evidence that the vaccinations B.A.B. received contained aluminum, glutamate, formaldehyde, or other unidentified pathogens. And even if B.A.B.'s vaccinations contained all of these alleged ingredients, Dr. Harum failed to show how they could have caused B.A.B. to develop encephalopathy. She cites two articles, one by Campbell et al. [45] and another by Giangaspero et al.[46] to support her generalizations, neither of which is relevant to B.A.B.'s case.

The Campbell researchers discuss the links between exposure to aluminum and the development of age-related neurological disorders, mainly focusing on Alzheimer's disease ("AD"). Pet'r's Ex. 28 at 40. Acknowledging that "epidemiological studies show that long-term

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[44] To support her assertion, Dr. Harum cited Campbell et al., "Chronic Exposure to Aluminum in Drinking Water Increases Inflammatory Parameters Selectively in the Brain," 75 J. NEUROSCI. RES. 565-72 (2004) (filed as Pet'r's Ex. 28 at 40-47).

[45] Campbell et al., supra note 42 at 565-72.

[46] Giangaspero et al., "Genotypes of Pestivirus RNA Detected in Live Virus Vaccines for Human Use," 63 J. VET. MED. SCI. 723-33 (2001) (abstract only filed as Pet'r's Ex. 28 at 134).

exposure to [aluminum] is necessary for adverse health effects," they exposed mice to levels of aluminum in drinking water for ten weeks, a period comparable to eight years for humans. Id. at 42. Although the activation of transcription factors (one of the initial steps of an inflammatory response) was found at the lowest levels of aluminum (comparable to levels found in certain areas of Canada), values associated with inflammation "reached significance only at the higher [levels of aluminum] exposures)." Id. at 43. The researchers cautioned that "it is possible that a threshold concentration of the metal has to be accumulated in the brain before an inflammatory response is even initiated." Id. at 43-44. Furthermore, they theorized that the "[i]nflammatory processes . . . within the aging brain . . . may provide the substrate upon which aluminum can act and thus *accelerate* the progression of age-related neurodegenerative disease." Id. at 44 (emphasis added). They concluded only that aluminum "could contribute to progressions of neurodegeneration." Id. at 40.

The authors of the Campbell study state, "Abnormal neurological symptoms have been observed in *several* patients receiving intramuscular injections of [aluminum]-containing vaccines," and "the [World Health Organization] Vaccine Safety Advisory Committee has recognized that there *may* be a subset of predisposed individuals who are sensitive to [aluminum]-containing adjuvant." Pet'r's Ex. 28 at 40 (internal citations omitted) (emphasis added). However, the authors do not reach any other conclusions. Vaccines are mentioned only briefly to provide support for the proposition that aluminum potassium sulfate is an adjuvant that "enhance[s] the systematic immune response." Id. at 44.

Dr. Harum does not suggest, and the record does not indicate, that B.A.B. was exposed to aluminum over a period of weeks or years. Moreover, B.A.B. is not elderly and Dr. Harum does not explain or suggest that B.A.B. is a "predisposed individual [] who [is] sensitive to [aluminum]-containing adjuvant." Pet'r's Ex. 28 at 40. While Dr. Harum postulates that the alleged high levels of aluminum found in the Proquad vaccines could have led B.A.B. to experience an "inflammatory event" such as an encephalopathy, the Campbell study and its findings are focused on long-term aluminum exposure through drinking water. Thus, the undersigned finds the Campbell article inapplicable to B.A.B.'s case.

Dr. Harum's cite to the Giangaspero study[47] is also not persuasive evidence that B.A.B. suffered an encephalopathy, and the undersigned struggles to see how it is relevant to B.A.B.'s case at all. The researchers tested 33 different live virus vaccines for human use[48] for the presence of pestivirus[49] and found that "[f]ive (13.1%) out of 38 tested samples were positive for

[47] The undersigned notes that petitioner did not file the full Giangaspero article but rather only a one page abstract. See Pet'r's Ex. 28 at 134.

[48] The vaccines tested included "29 monovalent vaccines against measles, mumps, rubella or polio, eight polyvalent vaccines against measles-mumps-rubella and one bacterial polyvalent vaccine against Streptococcus pneumoniae." Pet'r's Ex. 28 at 134.

[49] Pestivirus is "a genus of viruses of the family Flaviviridae comprising bovine diarrhea virus, hog cholera virus, and border disease virus of sheep. DORLAND'S at 1421-22.

pestivirus RNA." Pet'r's Ex. 28 at 134. According to the researchers, "[t]hese findings indicate that contamination by animal pestivirus may occur in biological products for human use." Id.

Presumably, petitioner introduced the Giangaspero article to show the potential for human vaccines to be contaminated. Assuming, *arguendo*, that contaminants were present in the vaccinations that B.A.B. received, Dr. Harum offers no explanation as to how these potential contaminants, including pestivirus, could have caused B.A.B. to develop encephalopathy. The abstract of the Giangaspero article that petitioner filed, without further explanation from Dr. Harum or petitioner, seems to be irrelevant to B.A.B.'s alleged development of encephalopathy.

Considering Dr. Harum's theory on the whole reveals a fundamental flaw: in her second report, she notes that there is evidence of loss of skills, language plateau, auditory awareness and visual attention after B.A.B.'s 12-month vaccinations. Pet'r's Ex. 26 at 1. However, the medical records demonstrate that B.A.B. did not begin to lose any skills until approximately 18 months of age, when petitioner first expressed concern to the pediatrician during an office visit on October 19, 2007. Pet'r's Ex. 6 at 4, 16. Moreover, at B.A.B.'s nine, 12, and 18-month well-child visits, he was noted to be a well-child and to have met all developmental milestones. Pet'r's Ex. 6 at 14-16. As for the abnormalities seen by Dr. Harum on September 26, 2008, when B.A.B. was 29 months old, including "reduced eye contact, limited cooperation with balance maneuvers, frequent tripping, [] intermittent toe walking … [and] oral aversion," Dr. Harum concedes that these physical findings "do not specifically relate to vaccination at 12 months of age, nor do they confirm regression of any sort." Pet'r's Ex. 29 at 7.

In addition to the issues with Dr. Harum's expert reports noted above, the undersigned notes several other problems. Dr. Harum fails to explain how subclinical encephalopathy would more likely result from the MMRV vaccine than from the viral illnesses accompanied by fever and other symptoms that B.A.B. experienced two to three months before the MMRV vaccine or four months after it. She makes vague references to vitamin, iron and folate deficiencies, "if present at the time of vaccine complications," as possible contributors to B.A.B.'s poor immune response. Pet'r's Ex. 19 at 1. However, the record reflects that B.A.B. did not experience such deficiencies, nor did he have a poor immune response to the MMRV vaccine. In fact, his pediatrician noted that he had a robust response to the measles portion. Dr. Harum refers to vaccine-caused seizures, but B.A.B. has never been diagnosed with a seizure disorder,[50] much less one temporally related to a vaccination.

Likewise, Dr. Harum makes vague references to markers of "oxidized DNA and reduced glutathione stores" and data which "suggest oxidized molecules are elevated" when B.A.B. was 29 months of age, but she does not explain how vaccines given at 12 months of age relate to these findings, or that these findings prove a vaccine related injury. See Pet'r's Ex. 29 at 6.

[50] While B.A.B.'s parents expressed concerns about seizures, no doctor ever diagnosed him as having had one. Dr. Harum also admits that B.A.B. never experienced even one seizure. Pet'r's Ex. 26 at 3. In her third report, Dr. Harum does clarify her position on several important issues. B.A.B.'s parents describe "spells," which were temporally associated with his 12 month vaccines. Pet'r's Ex. 29 at 7. Dr. Harum concedes, however, that these spells did not constitute seizures and that B.A.B. "clearly does not have a seizure disorder." Id.

b. Respondent's Expert, Dr. Gregory L. Holmes

Dr. Gregory Holmes testified on behalf of respondent. Dr. Holmes received his medical degree from Washington and Lee University, and he holds an honorary degree from Harvard. Resp's Ex. C at 1. He did an internship in pediatrics and completed his residency in pediatrics at the Yale University School of Medicine, after which he completed another residency in Neurology at the University of Virginia School of Medicine. Id. He is certified by the American Board of Pediatrics, the American Board of Psychiatry and Neurology with Special Competence in Child Neurology, and the American Board of Clinical Neurophysiology. Id. Dr. Holmes has also held various academic appointments, including his current positions as a professor of pediatrics at the University of Vermont College of Medicine and the professor and chair of the neurology and pediatrics department at Dartmouth Medical School. Id. at 2. He has received numerous academic and fellowship awards, and he has served as an editor for over a dozen pediatric and neurology medical journals. Id. at 5-7. In addition, he has published numerous medical articles on topics ranging from pediatric seizures to brain development and behavior. Id. at 39-65.

Dr. Holmes set forth a lengthy overview of B.A.B.'s medical history and clinical course, concluding that B.A.B. is an "eight year old boy with the diagnosis of autistic spectrum disorder (ASD) and language delay. His clinical history and neurological examination are quite consistent with this diagnosis." Resp's Ex. B at 7.[51]

As for Dr. Harum's conclusion that B.A.B. sustained encephalopathy, Dr. Holmes disagreed. Dr. Holmes defined encephalopathy as a "general term used to describe brain dysfunction." Resp's Ex. B at 7. He explains that the "clinical features" of encephalopathy include "a change in mental status, ranging from confusion to stupor or coma, alterations in muscle tone, abnormal movements or severe seizures." Id. Examples of encephalopathy set forth in the IOM include "encephalitis, meningitis, seizures and head trauma, none of which were suffered by [B.A.B.]." Id. Moreover, B.A.B.'s pediatrician saw him three times following the vaccinations at issue (July 14, August 15, and September 17), and in the records of those visits, "there is no mention of any behavior consistent with an encephalopathy." Id. at 7.

Dr. Holmes summarized, "The report of Dr. Harum does not dissuade me from my original opinion. There is no evidence in the medical record to indicate that [B.A.B.] suffered from an acute or subacute encephalopathy." Resp's Ex. Q at 4. He further stated, "[B.A.B.] has autistic spectrum disorder and the MMR vaccine has no bearing on his current clinical condition. Dr. Harum has provided no reliable evidence linking the MMR vaccine to autism." Id. As explained in further detail below, the undersigned finds Dr. Holmes' opinions to be more persuasive than those of Dr. Harum.

V. Encephalitis vs. Encephalopathy vs. Subacute/Subclinical Encephalopathy

To provide further clarity to the discussion of the Althen prongs, the undersigned first notes the differences between encephalitis and encephalopathy. Encephalitis is defined as

[51] It should be noted that respondent's Exhibit B was filed twice, once on December 17, 2014 (ECF No. 65) and again on January 7, 2015 (ECF No. 66).

"inflammation of the brain."[52] In contrast, encephalopathy is defined as "any degenerative disease of the brain."[53] Dr. Harum conflates these terms throughout her expert reports, despite the fact that they represent two distinct diagnoses. For example, in her first expert report, Dr. Harum states, ". . . there is a plausible body of evidence to associate MMR and other vaccines and *encephalitis* resulting in mental retardation and speech impairment." Pet'r's Ex. 19 at 2. From this assertion, she concludes that "one can postulate that there is a plausible association between MMR and other vaccines and *subacute encephalopathy* associated with developmental regression." Id. (emphasis added). While Dr. Harum is correct that the MMR vaccination has been known to cause acute encephalopathy in children five to 15 days post vaccination,[54] it is incorrect to assume that therefore there is a relationship between MMRV and "subtle" or subacute encephalopathy, as described by. Dr. Harum.[55]

While Dr. Harum eventually concludes that B.A.B. did not suffer from encephalitis and only from encephalopathy, the central component of her theory is that B.A.B. suffered from a "subacute" or "subclinical" encephalopathy. Dr. Harum describes B.A.B.'s condition as subacute or subclinical "because it was not acute and was subtle enough for parents to recognize, but not obvious enough for the pediatrician to be concerned about or to inhibit ridicule of Mom's internet research." Pet'r's Ex. 29 at 4. She believes that B.A.B.'s "developmental plateau . . . . suggests a subacute encephalopathy." Pet'r's Ex. 26 at 4. Despite the fact that none of B.A.B.'s treating physicians ever suggested a diagnosis of encephalopathy, Dr. Harum presumes that B.A.B. suffered "subclinical encephalopathy," which went unnoticed by his physicians during numerous doctor's appointments over a period of several months.

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[52] DORLAND'S at 612.

[53] DORLAND'S at 614.

[54] While petitioner has not alleged that B.A.B. suffered a Table encephalopathy, it is important to note that "encephalopathy" does exist as a Table injury for several vaccinations, including pertussis or measles containing vaccines. However, the Table specifies a lengthy and precise definition of an "encephalopathy." See 42 C.F.R. § 100.3(b)(2). To summarize, the regulation requires a "significantly decreased level of consciousness" that lasts at least 24 hours, which was not seen in B.A.B.'s case. As former Chief Special Master Campbell-Smith noted in Waddell v. Sec'y of Health & Human Servs., No. 10-316V, 2012 WL 4829291, at *6 (Fed. Cl. Spec. Mstr. Sept. 19, 2012), the symptoms of a Table Injury encephalopathy are not "subtle."

[55] It should also be noted that Dr. Harum does not give a citation to a definition of subacute or subclinical encephalopathy. Furthermore, Dr. Holmes noted:

> The pathophysiological mechanisms by which such a "subtle encephalopathy" could result in autism spectrum disorder is not discussed by Dr. Harum. The term "subtle encephalopathy" is not a medically recognized entity and it is difficult to understand how such a subtle process could result in harm. It is highly unlikely that a vaccine-induced encephalopathy could be so subtle that it was not noted by the pediatrician.

Resp's Ex. Q at 3.

VI. Petitioner has Failed the Althen Test

In Althen, the United States Court of Appeals for the Federal Circuit discussed the issue of "causation-in-fact" in Vaccine Act cases. The court stated:

> [Petitioner's] burden is to show by preponderant evidence that the vaccination brought about [the child's] injury by providing: (1) a medical theory causally connecting the vaccination and the injury; (2) a logical sequence of cause and effect showing that the vaccination was the reason for the injury; and (3) a showing of a proximate temporal relationship between the vaccination and injury. If [petitioner] satisfies this burden, she is entitled to recover unless the [government] shows, also by a preponderance of the evidence, that the injury was in fact caused by factors unrelated to the vaccine.

Althen v. Sec'y of Health & Human Servs., 418 F.3d 1274, 1278 (Fed. Cir. 2005) (internal citations and quotations omitted). In the pages above, the undersigned has provided a detailed explanation of how petitioner has failed to demonstrate preponderant evidence of "causation-in-fact." The next section shows how that analysis fits within the three prongs of the Althen test. For the reasons set forth below, the undersigned finds that petitioner has failed to satisfy the Althen test and is therefore not entitled to compensation.

A. Althen Prong One: Lack of a Reliable Medical Theory

As discussed below, petitioner has failed Prong One of Althen for three reasons. First, the record lacks evidence to show that Proquad can cause encephalopathy or sub-clinical encephalopathy, as described by Dr. Harum. Second, Dr. Harum mischaracterizes B.A.B.'s diagnosis as an encephalopathy despite the fact that he was clearly diagnosed with ASD. Finally, Dr. Harum's medical theory bears a striking resemblance to the theory proposed by Dr. Marcel Kinsbourne during the OAP, which was found unpersuasive in all three test cases.

a. Lack of Evidence Demonstrating that Proquad can Cause Encephalopathy or "SubClinical" Encephalopathy

Prong One of Althen requires petitioner to show by preponderant evidence a medical theory of how the MMRV vaccination B.A.B. received on April 18, 2007, can cause encephalopathy. As discussed above, however, Dr. Harum has not demonstrated that the Proquad vaccine can cause encephalopathy. Instead, Dr. Harum has conflated the definitions of encephalitis and encephalopathy and has presented a number of claims and statements in her expert reports which lack foundation.

b. Mischaracterization of B.A.B.'s ASD Diagnosis as a "Subclinical" Encephalopathy

In denying petitioner's earlier motion for interim fees and costs, former Chief Special Master Vowell noted that after the OAP proceedings, some petitioners chose, in spite of an ASD diagnosis, to pursue their cases by characterizing their children's injuries as "encephalopathy" or seizure disorders accompanied by developmental delay, among others. Former Chief Special Master Vowell observed that petitioners often chose to amend their theories "based on a belief

that claims alleging autism [would] not be compensated, but that claims alleging these other conditions may be compensated." Fester v. Sec'y of Health & Human Servs., No. 10-243V, 2013 WL 5367670, at 1 n.5 (Fed. Cl. Spec. Mstr. Aug. 27, 2013). She further stated, "[R]e-characterizing a condition as an "encephalopathy"—a term that can encompass conditions ranging from intoxication to a coma—when another diagnosis is more specific and appropriate does little to advance a vaccine injury claim." Id.[56]

Not only does Dr. Harum conflate encephalopathy and encephalitis, but she has offered no support for her proposed theories of causation. Although not required to provide medical literature to support her theory, the medical literature Dr. Harum has cited provides no support. See Andreu ex rel. Andreu v. Sec'y of Health & Human Servs., 569 F.3d. 1367, 1378-79 (Fed. Cir. 2009) (explaining that "requiring 'objective confirmation' in the medical literature prevents the use of circumstantial evidence … and negates the system created by Congress through the Vaccine Act" (internal citations omitted)). However, Dr. Harum is still required to prove her theory by preponderant evidence, which she has failed to do.

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[56] Though the issue of causation was not adjudicated, former Chief Special Master Campbell-Smith explained in Poling v. Sec'y of Health & Human Servs., No. 02-1466V, 2011 WL 678559 (Fed. Cl. Spec. Mstr. Jan. 28, 2011), that petitioners received compensation because the child suffered a Table Injury, specifically a Table encephalopathy, and not because respondent conceded or the special master found that the child's ASD was either caused or significantly aggravated by a vaccination. Similarly, in Wright v. Sec'y of Health & Human Servs., No.12-423V, 2015 WL 6665600, at *30 (Fed. Cl. Spec. Mstr. Sept. 21, 2015), former Chief Special Master Vowell found that a child who was later found to have ASD suffered a Table Injury (encephalopathy) after receiving a vaccination. In finding that petitioner was entitled to compensation, however, Special Master Vowell emphasized that she was not finding that the child's ASD was "caused-in-fact" by the vaccination, even remarking that petitioner's medical theory of causation was "absurd." See Sturdivant, 2016 WL 552529, at *5, note 4.

As Special Master Hastings has noted, the compensation of the petitioners in Poling and Wright "does *not* afford any support to the notion that vaccinations can contribute to the *causation* of autism …. Congress forthrightly acknowledged that the Table Injury presumptions would result in compensation for some injuries that were *not*, in fact, truly vaccine caused." Sturdivant v. Sec'y of Health & Human Servs., No. 07-788V, http://www.help.senate.gov/hearings/s-2700-s185-s2713-s_-nih-strategic-plan-and-inclusion-in-clinical-research-and-s_-promoting-biomedical-research-and-public-health-for-patients-act, 2016 WL 552529, at *5 n.4 (Fed. Cl. Spec. Mstr. Jan 21, 2016)(emphasis in original). In this case, petitioner has made clear that although B.A.B. suffers from ASD, her medical theory is that B.A.B developed encephalopathy from the Proquad vaccination, which subsequently caused him to develop a serious brain injury and developmental delay. Whether characterized as autism or encephalopathy, petitioner still has to prove that B.A.B.'s injury was "caused-in-fact" by the Proquad vaccination, and this she has not done.

c. Dr. Harum's Medical Theory of Causation is Similar to the Earlier Proposed Theory of Dr. Marcel Kinsbourne

In addition to being unable to articulate a reliable medical theory tending to show that MMRV can cause encephalopathy, the undersigned further notes that certain parts of Dr. Harum's theory are remarkably similar to the theory and testimony of Dr. Kinsbourne, as given in Snyder, 2009 WL 332044, which was part of the OAP. Dr. Kinsbourne was the leading expert witness for petitioners in the Theory 1 test cases.[57] "Dr. Kinsbourne's role was to provide the theory or theories to explain how measles virus could, directly or indirectly, cause at least some cases of ASD." Snyder, 2009 WL 332044, at *87. Dr. Kinsbourne offered two overarching theories to explain how the measles virus could lead to the development of ASD. Id. Special Master Vowell explained, "The first theory was that the virus caused an inflammatory process in the brain leading to an encephalopathy. The second theory, built in some measure on the first, relied on persistent measles virus causing inflammatory damage to cells, leading to an imbalance in the excitation-inhibition chemicals in the brain." Id.

Dr. Kinsbourne's theory was divided into three core stages. Stage One occurred as the brain's innate immune system caused neuroinflammation, mediated by microglial activation, which in turn caused the release of cytokines and damaged astrocytes. As a result of damage to the astrocytes, brain glutamate levels rose. Snyder, 2009 WL 332044, at *87. In Stage Two, the excess glutamate in the brain caused over-arousal or over-activation of the brain, which then caused the potential for seizures and neuronal death as the result of excitotoxicity. Id. In Stage Three, Dr. Kinsbourne hypothesized that "[n]eural activation and over-arousal could account for autistic behavior." Id.

Dr. Harum's theory bears a striking resemblance to Dr. Kinsbourne's three tiered theory, which was found to contain no persuasive evidence that the MMR vaccine contributed in any way to cause ASD. For example, Dr. Harum opines:

> These mechanisms of injury contribute to [B.A.B.'s] autism spectrum condition through an increase in *brain inflammation*, a pathophysiologic mechanism well described in spectrum disorders. This mechanism converges onto the more recently described neuronal pathways and signaling systems identified as

[57] The Petitioners' Steering Committee ("PSC"), formed in 2002 by petitioners' attorneys in the Vaccine Program, presented two separate theories in the OAP regarding how vaccines cause ASD. "The first theory alleged that the *measles* portion of the measles, mumps, rubella ("MMR") vaccine could cause ASDs. That theory was presented in three separate Program test cases during several weeks of trial in 2007." Sturdivant, 2016 WL 552529, at *3 (emphasis in original). In all those cases, the special masters rejected petitioners' theory of causation. See Cedillo, 2009 WL 331968; Hazlehurst v. Sec'y of Health & Human Servs., 03-645V, 2009 WL 332306 (Fed. Cl. Spec. Mstr. Feb. 12, 2009), aff'd, 88 Fed. Cl. 473 (2009), aff'd, 604 F.3d 1343 (Fed. Cir. 2010); Snyder, 2009 WL 332044.

impaired in autism by virtue of *over activity of the glutamate receptors and excitetoxicity [sic]* to developing synapses.

Pet'r's Ex. 26 at 3 (emphasis added). Dr. Harum also states, "The evidence that immune deregulation is associated with an autism diagnosis lies in the landmark data that points toward *chronic inflammation* in autism. The [Vargas] authors demonstrated long-term *microglial activation* and *abnormally high cytokine levels*." Id. at 2 (emphasis added).

It appears from these and other similar statements that Dr. Harum simply borrows and enhances certain aspects of Dr. Kinsbourne's earlier theory in an effort to opine that the Proquad vaccine could cause encephalopathy and eventually lead to neuronal damage. However, Dr. Harum, like Dr. Kinsbourne, has failed to demonstrate a reliable theory demonstrating that MMR can cause subacute encephalopathy. Dr. Kinsbourne's theory was ultimately rejected in the three tests cases. See Cedillo, 2009 WL 331968; Hazlehurst, 2009 WL 332306; Snyder, 2009 WL 332044. So too, the undersigned rejects Dr. Harum's similar reasoning in this case.

For all of the reasons stated above, petitioner has failed to provide evidence of causation sufficient to meet Prong One of Althen.

B. **Althen Prong Two: Lack of a Logical Sequence of Cause and Effect**

Althen Prong Two requires petitioner to show by preponderant evidence a logical sequence of cause and effect to explain how the MMRV vaccination caused B.A.B. to develop encephalopathy. As explained above, the medical records are devoid of any evidence that B.A.B. ever developed or was diagnosed with encephalopathy. Moreover, Dr. Harum's three medical expert reports do not clearly or logically delineate facts to show that B.A.B. developed encephalopathy, much less that the MMRV vaccine actually caused this alleged encephalopathy.

Dr. Harum also relies upon factual inaccuracies and medical presumptions which are not supported by B.A.B.'s medical records. For example, the first step of Dr. Harum's theory of causation is that B.A.B. suffered from roseola infantum at the age of nine months. See Pet'r's Ex. 29 at 8; Pet'r's Ex. 26 at 3. However, it is clear from the record that B.A.B. was not diagnosed with roseola infantum until the age of 22 months, which was 10 months after he received the Proquad vaccine. Dr. Harum did not become B.A.B.'s treating physician until he was 29 months old, and thus her medical opinion is based on B.A.B.'s pediatric records. These records do not indicate that B.A.B. suffered from roseola infantum prior to the age of 22 months, and B.A.B.'s treating physicians did not ever consider the diagnosis until that time.

Additionally, despite Dr. Harum's contention that the vaccines were given in proximity to a "significant encephalitic virus infection" or a "mild and temporary encephalitis," there are no records or notes from any of B.A.B.'s doctors regarding a condition or diagnosis, despite the fact that B.A.B. visited his pediatrician a number of times while he ostensibly suffered from the condition. Pet'r's Ex. 26 at 3; Pet'r's Ex. 29 at 3. The assertion that B.A.B. suffered from a subacute encephalopathy that went undetected and untreated by pediatricians for several months is unfounded. The undersigned further notes that no previous Program cases have awarded compensation to petitioners based on a diagnosis of subacute or subclinical encephalopathy.

Dr. Harum suggests that B.A.B.'s lab results are evidence of an apparent, undetected encephalopathy. She points to B.A.B.'s granular leukocyte count, elevated NSE levels, abnormal IgG count, and Il-2 Receptor Alpha elevation to support these assertions. She follows her discussion of these lab results with citations to a number of medical articles that supposedly support her contention that B.A.B. suffered from encephalopathy. However, Dr. Harum does not actually explain how their findings are applicable to B.A.B.'s case and/or discuss how they provide evidence that he suffered an encephalopathy.

Upon further examination of the medical articles cited by Dr. Harum, most are irrelevant to B.A.B.'s diagnosis of ASD and a few actually contradict her opinions altogether. For example, as discussed above, Dr. Harum's citation to the Berger[58] study actually disproves her theory that B.A.B.'s elevated NSE levels point to "ongoing neuronal injury" and encephalopathy. According to the Berger study, if B.A.B. had suffered from an inflicted TBI, one would expect his NSE levels to peak up to eight days after the injury. The study's findings suggest that B.A.B.'s elevated NSE levels on September 29, 2008, were the result of a much more recent injury than his April 18, 2007 Proquad vaccination.

Dr. Harum's second and third medical expert reports adapt a "kitchen sink approach" of including all potential medical theories, such as the theory that "the load of aluminum, glutamate, formaldehyde and unidentified pathogens" presumably contributed to B.A.B.'s development of encephalopathy. See Pet'r's Ex. 26 at 3; Pet'r's Ex. 29 at 8. Dr. Harum seems to suggest that the amount of aluminum found in the PedVax and the Proquad vaccines contributed to the alleged development of encephalopathy. However, petitioner contends that the Proquad vaccine, not PedVax, caused B.A.B.'s encephalopathy. Petition at Preamble. Moreover, the articles cited by Dr. Harum do not support her assertion that foreign contaminants found in vaccines contribute to neurological injuries. Dr. Harum merely cites these studies and does not provide any explanation as to how or why they are applicable to B.A.B.'s case.

Thus, petitioner has failed to provide preponderant evidence of actual causation under Althen Prong Two.

C. **Althen Prong Three: Lack of a Temporal Relationship**

Under Prong Three of Althen, petitioner must show by preponderant evidence B.A.B.'s encephalopathy occurred within a time frame that is medically appropriate for the alleged mechanism of harm. See Pafford, 451 F.3d 1352, 1358 (Fed. Cir. 2006) ("Evidence demonstrating petitioner's injury occurred within a medically acceptable time frame bolsters a link between the injury alleged and the vaccination at issue under the 'but-for' prong of the causation analysis."). As the undersigned has already explained, Dr. Harum did not offer sufficient evidence tending to show that the Proquad vaccination causes encephalopathy, much less that B.A.B. developed an encephalopathy from the Proquad vaccination. It follows, then, that petitioner cannot prove Prong Three of Althen.

[58] Berger et al., 109 PEDIATRICS E31 (2002) (filed as Pet'r's Ex 27 at 1-8).

Even if Dr. Harum had shown that B.A.B. developed encephalopathy after receiving the MMRV vaccination, Dr. Harum does not provide preponderant evidence of a temporal relationship between the vaccine and B.A.B.'s alleged encephalopathy. Dr. Harum does not address the issue of a proximate temporal relationship between Proquad and encephalopathy other than to note the IOM's conclusion that the measles vaccine "can cause measles inclusion body *encephalitis* [not encephalopathy] due to the vaccine strain of measles, after administration of the measles vaccine, particularly in immune compromised individuals up to nine months after the vaccine administration." IOM at 110 (emphasis added); see also Pet'r's Ex. 26 at 1. Apart from this assertion, Dr. Harum does not offer any medical theory showing a proximate temporal relationship between B.A.B.'s vaccination and his alleged encephalopathy. Therefore, petitioner has failed to prove Prong Three of Althen.

VII. Conclusion

For the reasons discussed above, the undersigned finds that petitioner has not established entitlement to compensation and her petition must be dismissed. **Therefore, this case is dismissed for insufficient proof. The Clerk shall enter judgment accordingly.**

IT IS SO ORDERED.

s/ Nora Beth Dorsey
Nora Beth Dorsey
Chief Special Master